SOIL AND GROUNDWATER REMEDIATION GUIDELINES FOR DIETHYELENE GLYCOL AND TRIETHYLENE GLYCOL

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1. INTRODUCTION

Glycols are polyhydric alcohols, that is, aliphatic compounds with two or more hydroxyl (-OH) groups per molecule. Glycols have a wide range of uses including chemical feedstocks, solvents, and antifreeze. In addition, glycols are used in the dehydration of natural gas streams. Water in natural gas can cause operational problems in the transmission and processing of the gas (Sorensen et al., 2000), and thus gas dehydration units are ubiquitous at gas well sites and processing facilities. The most common dehydrating process used in the gas industry is the glycol absorption/stripping process (Katz and Lee, 1990). It is estimated that about 100,000 glycol dehydrating units exist worldwide (Grizzle, 1993).

The three glycols originally considered for soil and groundwater remediation guideline development were diethylene glycol (DEG), triethylene glycol (TEG), and tetraethylene glycol (TREG), which are formed by creating ether linkages between 2, 3, and 4 units of ethylene glycol (EG), respectively. Synonyms for these compounds are provided in Table 1. All three compounds have been used in glycol dehydration units. However, due to the lack of published toxicological information on TREG, and the fact that the use of this compound in glycol dehydration units is uncommon, only DEG and TEG were carried forward to the guideline development stage. The limited background data that were found concerning TREG are retained in this document for completeness.

For convenience, DEG, TEG, and TREG are collectively referred to in this document as "the Glycols". No soil or groundwater remediation guidelines have been published to date for any of the Glycols by either Alberta Environment (AENV) or the Canadian Council of Ministers of the Environment (CCME). This document develops proposed soil and groundwater remediation guidelines for DEG and TEG consistent with the Alberta Environment (AENV, 2009a) protocol.

Appendices A, B and C provide degradation and toxicological data specific to each of the three compounds, and include tables designated "Table A-1", "Table B-2", etc. Please refer to the appropriate appendices when reference is made to the corresponding table.

2. BACKGROUND INFORMATION

2.1 Chemical and Physical Properties

Chemical and physical properties of the Glycols are summarized in Table 2. The Glycols are polar organic compounds that are miscible with water. They have boiling points ranging from 245-314 °C and thus have negligible vapour pressures at typical environmental temperatures. The dimensionless Henry's law constant reported for TEG is 5.3×10^{-9} indicating that TEG will not volatilize when dissolved in water. DEG is assumed to have a dimensionless Henry's law constant close to zero. The negative log K_{oc} and K_{ow} values indicate that the Glycols would not be expected to be retarded in soil-groundwater systems or bioaccumulate in mammals.

2.2 Analytical Methods

One of the principal reference sources for analytical methods for water, soils, and other materials is the United States Environmental Protection Agency (U.S. EPA) Document SW-846: "*Test Methods for Evaluating Solid Wastes – Physical/Chemical Methods*" (U.S. EPA 2007d). The information below was summarized from this document.

One Alberta environmental laboratory analyzes glycols in soil samples using a modified version of U.S. EPA Method 3550A – "Ultrasonic Extraction", followed by U.S. EPA Method 8000A – "Determinative Chromatographic Separations". U.S. EPA (2007d) recommends method 8430 – "Analysis of bis(2-chloroethyl) ether and hydrolysis products by direct aqueous injection GC/FT-IR" for determination of EG and DEG.

Mrklas et al. (2003) developed a method for the analysis of TEG in water samples using ion exclusion chromatography with pulsed amperometric detection. Mrklas et al. (2004) used centrifugation to separate the supernatant prior to using the above technique on a soil/groundwater slurry.

2.3 Production and Uses

Major uses of DEG and TEG are shown in Figure 1.

Overview of Glycol Usage in Gas Dehydration

All three of the Glycols are used in natural gas dehydration. The following changes occur as the glycol chain length increases from DEG through TEG to TREG and are the main factors controlling the selection of the glycol compounds used in oilfield dehydration operations:

- increasing thermal stability;
- decreasing capacity for absorbing water; and,
- increasing cost.

DEG was commonly used for dehydration of natural gas prior to 1950 (Pearce, 1982), but is now used on relatively few units. Since 1950, TEG has been the most commonly used glycol for natural gas dehydration (Pearce, 1982). It has been estimated that TEG is currently used in 95% of glycol dehydration units (Thompson et al., 1993). TREG has the highest thermal stability and the lowest volatility of the glycols used for the dehydration of natural gas. However, because of its relatively high cost, it is generally used only in specialized cases (Sorensen et al., 1996). Small dehydration units are commonly located at wellsites, while larger glycol dehydrators may be present at natural gas processing plants.

The production and other uses of each of these compounds are discussed individually below.

DEG

DEG is a coproduct (9-10%) in the commercial synthesis of ethylene glycol by the hydrolysis of ethylene oxide. The quantity of co-product DEG produced exceeds demand for this chemical. DEG was first marketed by Union Carbide in 1928. The Dow Chemical Company remain a major supplier of this chemical. The global 1993 capacity for DEG production was estimated to be 359,000 ton/yr (Kirk-Othmer, 1999).

Major uses of DEG are summarized in Figure 1, and are as follows (Kirk-Othmer, 1999):

Natural Gas Dehydration	6%
Polyester Resins	45%
Antifreeze	14%
Manufacture of TEG	12%
Manufacture of Morpholine	10%
Miscellaneous*	13%

* Miscellaneous uses of DEG include plasticizers for paper, fiber finishes, compatibilizers for dye and printing ink components, latex paint antifreeze, and lubricants (Kirk-Othmer, 1999).

TEG

TEG is a minor coproduct in the commercial synthesis of ethylene glycol by the hydrolysis of ethylene oxide. However, the quantities produced in this process are not sufficient to satisfy demand, and additional TEG is produced by the reaction of ethylene oxide with DEG. The Dow Chemical Company is a major supplier of this chemical.

Major uses of TEG are summarized in Figure 1, and are as follows (Kirk-Othmer, 1999):

Natural Gas Dehydration	45%
Vinyl Plasticizer	13%
Solvent	11%
Manufacture of Ester Derivatives	12%
Miscellaneous	19%

TREG

TREG is produced by reacting lower molecular mass glycols (EG, DEG, TEG) with ethylene oxide. TREG is used in specialist natural gas dehydration applications. Although it is less hygroscopic than the lower members of the glycol series, it has a greater thermal stability. Other uses of TREG include a plasticizing agent for a variety of materials and an extraction solvent for benzene, toluene, and xylenes. No information was available on the relative amounts of TREG used in these various applications (Kirk-Othmer, 1999).

2.4 Sources and Emissions

Glycols in dehydrating units can potentially be released to the environment via spills, leaks, foaming events, or poor disposal of waste during changeover of units (Sorensen et al., 2000). Changeover refers to the process of replacing spent glycol with fresh, replacing filters, and cleaning/servicing the units. Glycols in gas dehydrating units are referred to as “raw”, “rich”, or “lean”. Raw glycol refers to the fresh compound prior to use in a dehydrating unit. Rich glycol is glycol that has passed through the absorber and is enriched with water and possibly other polar chemicals from the gas stream. Lean glycol is glycol that has been regenerated in the boiler to remove the water and some of the other polar compounds.

A foaming event in a glycol-based dehydration unit is typically caused by the presence of hydrocarbons in the glycol stream, excessive use of additives, or high concentrations of degradation products (Sorensen et al., 2000). Foaming events can release a mixture of lean and rich glycols to the environment.

Changeover (periodic maintenance and cleaning) operations are also likely sources of fresh and spent glycols. The frequency of changeover operations varies from unit to unit and operator to operator, but they typically occur on a seasonal basis, though some units may go years between changeover events (Sorensen et al., 1996). The spent glycol solutions collected during the changeover process are typically managed by disposal into process waste pits. Historically, these pits may not have been lined. Leaking process waste pits have been the most common, and largest source of groundwater contamination at gas processing plants (Sorensen et al., 1996).

Leaks from dehydration units can occur, and may release significant amounts of rich or lean glycol into the environment, depending on the severity and duration of the leak. Catch basins may be placed under the valves and spigots at newer units to minimize the likelihood of leaks reaching the environment, but some older facilities may not use any management techniques (Sorensen et al., 1996).

Release of glycol to the atmosphere via regenerator off-gas is likely not a concern, based on the low vapour pressure of glycol (TEG has a vapour pressure of <1 mm Hg at 100 °C). However, note that the release of other compounds in the regenerator off-gas, particularly benzene, is a significant concern at some glycol dehydrator installations.

Glycol dehydration wastes may contain a number of co-contaminants, which are chemicals removed from the gas stream or glycol degradation products. Myerski et al. (1993) sampled spent glycols from storage tanks and dehydration units, and found that the concentrations of benzene in these waste streams can be as high as 110 mg/kg in TCLP[1] analysis. Sorensen et al. (2000) analyzed 29 samples of raw, rich, and lean glycols from gas processing facilities across north America, and found that rich glycols can contain relatively high levels of benzene, toluene, ethylbenzene and xylenes (BTEX), and potentially significant concentrations of naphthalene and other polycyclic aromatic hydrocarbons (PAHs). Lean glycols typically contained much lower concentrations of BTEX, and similar concentrations of naphthalene and other PAHs to rich glycols.

Glycols also have non-oilfield uses. The industrial uses (resins, plasticizers, manufacture of other chemicals) could potentially result in releases to the environment, but this is likely a concern only at a small number of facilities where these chemicals are manufactured or used for manufacturing other chemicals. Significant quantities of glycols can be used in aircraft de-icing operations. Aircraft de-icing fluids are typically composed primarily of ethylene glycol and/or propylene glycol, but may contain a small proportion of DEG (Sorensen et al., 1996).

2.5 Distribution in the Environment

No information was found that would indicate DEG, TEG, or TREG occur naturally in the environment. Accordingly, their distribution in the environment is expected to be strongly biased towards facilities where these compounds are produced or used. The number of facilities where the Glycols are used is significant. In the U.S., national surveys of occupational hazards were carried out in 1974 and 1983. The 1983 survey (NIOSH, 1983) indicated that the number of facilities where these glycols was used and the number of employees exposed to each was:

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[1] Toxicity Characteristic Leaching Procedure (TCLP)

Glycol	*Number of Facilities*	*Number of Employees Exposed*
DEG	55,518	890,145
TEG	23,174	233,613
TREG	3,704	55,282

The physical and chemical properties of these glycols (Table 2) control the environmental media in which they are likely to be found. All three glycols have very low vapour pressures, and accordingly, their presence in the atmosphere will not be significant. All the Glycols could potentially be present in soil, groundwater, and/or surface water in the vicinity of facilities where they are used.

Glycol releases from oil and gas facilities can occur as a result of leaks from operating equipment, or through the improper disposal of wastes when glycol-using facilities are maintained.

Spills and releases of DEG, TEG, and TREG at gas plants are remediated where possible. In Alberta, frequency of spill reporting and concentrations of DEG are generally higher than TEG and TREG, with TREG typically having concentrations less than 10 mg/kg or non-detectable concentrations.

2.6 Human Exposure

Based on the physical and chemical properties of the Glycols, human exposure can occur via soil and water, but is not likely via the atmosphere, due to the negligible vapour pressure of the Glycols (Table 2). The potential, and very unusual, exception might be workers who could be chronically exposed to glycol fogging agents in theatre productions. Exposure via food and consumer products is possible for all the Glycols - the European Community have developed tolerable daily intakes for each of the Glycols to account for their possible presence in food-grade plastics, (European Commission, 2003). In addition, DEG is used in automobile antifreeze and brake fluids.

No regulatory estimates of the daily human exposure to the Glycols are available; therefore, in the absence of supporting information, the human estimated daily intake and the ambient air concentration and background soil concentration are assumed to be zero in areas isolated from facilities where the Glycols are used.

2.7 Existing Criteria, Guidelines and Standards

Only very limited information was found concerning guidelines, criteria, and standards for these chemicals.

Canadian Federal

CCME (1999 and updates) provides soil quality guidelines for ethylene glycol (960 mg/kg, based on the groundwater check for aquatic life). No CCME soil quality guidelines have been developed for DEG, TEG, or TREG. CCME (1999 and updates) provides water quality guidelines for the protection of freshwater aquatic life for ethylene glycol (192 mg/L) and propylene glycol (500 mg/L). Earlier CCME documents did have a water quality guideline for DEG, but this was rescinded in 1997, based on a lack of sufficient information. CCME water quality guidelines have not been developed for TEG or TREG.

Health Canada (2007) does not include any glycols in its "Guidelines for Canadian Drinking Water Quality". Health Canada (2004) does not publish Tolerable Daily Intakes for any glycols.

Canadian Provincial

The Ontario Ministry of Environment (OMEE, 1994) published an Interim Provincial Water Quality Objective (Interim PWQO) for DEG (11 mg/L). They also have PWQOs for EG, 1,2-propylene glycol and 1,3-propylene glycol. These Interim PWQOs are based on protection of all forms and life-stages of aquatic life for exposure over an extended period. No other provincial soil or water quality guidelines for DEG, TEG, or TREG were found.

US Federal

The U.S. EPA (2006, 2007c) does not publish a water quality guideline for DEG, TEG, or TREG protective of aquatic life, or Maximum Contaminant Levels (MCLs) for DEG, TEG, or TREG in drinking water. Neither DEG, TEG, nor TREG are included in the list of chemicals for which the U.S. EPA publishes Ecological Soil Screening Levels (EcoSSLs). No toxicological information is available on the U.S. EPA (2007a) IRIS database for DEG, TEG, or TREG.

US State

No criteria, guidelines, or standards were found in a limited search of U.S. state information.

Europe

The Dutch Ministry of the Environment (VROM, 2000) have published "Indicative Levels for Serious Contamination" for DEG (270 mg/kg in soil, and 13 mg/L in groundwater). VROM (2000) also included a DEG value for Human Maximum Permissible Risk (MPR) level of 0.4 mg/kg bw/day and an ecological HC50 of 480 mg/kg. HC50 is the hazardous concentration 50%, *i.e.*, the concentration at which 50% of the species and processes in an ecosystem are completely protected. VROM (2000) also have levels for EG, but not TEG, or TREG.

The United Kingdom Environment Agency develops Soil Guideline Values (SGVs) under its Contaminated Land Exposure Assessment (CLEA) program. Glycols have been identified for consideration, but no guidelines have been developed to date.

No other European guidelines for DEG, TEG, or TREG in soil or groundwater were found.

Australia and New Zealand

Australia and New Zealand have a collaborative set of water quality guidelines protective of aquatic uses (ANZECC, 2000). These guidelines do not include values for DEG, TEG, or TREG. No Australian drinking water guideline has been set for DEG, TEG, or TREG (NHMRC, 1996).

Global

The World Health Organization (WHO, 2004) does not include glycols in its "Guidelines for Drinking Water Quality, Third Edition".

Occupational Exposure Limits

The following occupational inhalation exposure limits for DEG are listed by NIOSH (2003):

Denmark:	time-weighted average 2.5 ppm (11 mg/m^3)
Poland:	MAC (time-weighted average) 10 mg/m^3
Russia:	short term exposure limit 10 mg/m^3
Sweden:	time-weighted average 10 ppm (45 mg/m^3) short term exposure limit 20 ppm (90 mg/m^3)
United Kingdom:	time-weighted average 23 ppm (101 mg/m^3)

3. ENVIRONMENTAL FATE AND BEHAVIOUR

3.1 Adsorption and Mobility

DEG and TEG have negative log K_{ow} (octanol-water partition coefficient) and log K_{oc} (organic carbon-water partition coefficient) values (Table 2), indicating that they partition to water in preference to a non-polar solvent or to soil organic carbon. Therefore, their mobility in the subsurface is unlikely to be limited by sorption to organic carbon.

No experimental data for the soil-water partition coefficient (K_d) of the Glycols were available. The Glycols are not significantly ionized at environmental pH values, as indicated by their high pKa values (14.5 for TEG, Table 2; 14.2 for EG; DEG assumed to be similar). Thus, interactions between ionized forms and charged clay surfaces can be ruled out. However, the Glycols are polar molecules and, as such, weak interactions with charged surfaces of clay minerals are expected. However, analytical techniques using an aqueous extraction appear to recover the Glycols quantitatively from soil samples, and thus it would appear that the binding of the Glycols to soils is minimal and the effective K_d of the Glycols is very small.

The values of K_d used in this document to assist in predicting the mobility of the Glycols in the subsurface were 9.0×10^{-5} L/kg for DEG and 2.6×10^{-5} L/kg for TEG, and were calculated by multiplying the Koc values in Table 3 by the assumed fraction of organic carbon in soil (0.005, Table 5).

3.2 Aqueous-Phase Solubility

The Glycols are all considered miscible with water (Table 2). Accordingly, the mobility of these compounds in the subsurface will not be limited by solubility.

3.3 Co-Solvency of Glycols and Hydrocarbons

Glycol dehydrators will remove lighter aromatic hydrocarbons (*e.g.*, BTEX, naphthalene, and their derivatives) from the gas stream where these compounds are present. Accordingly, releases of glycols to the environment may occur at the same location as releases of hydrocarbons. Sorensen et al. (2000) investigated the possible co-solvency of glycols and hydrocarbons, by investigating whether the addition of TEG could enhance the mobility of BTEX and naphthalene by reducing their soil/water partitioning coefficient (K_d). Their results varied with soil type and organic carbon content, but in several cases they were able to demonstrate a significant and sometimes dramatic reduction in the K_d values for BTEX, and particularly naphthalene, in the presence of glycols. For example, the presence of a 40% solution of TEG reduced the K_d of naphthalene in an Alberta Till soil from 6.3 to <0.5 (units unspecified, but assumed to be L/kg).

3.4 Leaching and Lateral Movement

As noted above, the movement of the Glycols in the subsurface is not likely to be limited by either adsorption to organic carbon or solubility. The degree of sorption, if any, to clay minerals is not known. Consequently, leaching and lateral movement may be potentially significant factors in the subsurface transport of the Glycols.

3.5 Biodegradation

Although glycol releases at gas plants, airports, and other facilities are well documented, glycol plumes are not typically extensive in fine-grained Alberta soils, suggesting that biodegradation, and possibly other sorption and/or attenuation processes are active.

3.5.1 Degradation Pathways

Aerobic biodegradation of ethylene glycol has been suggested (e.g., Gonzalez et al., 1972; Caskey and Tabor, 1981) to occur via the pathway:

ethylene glycol → glycollate → glyoxylate → tartronic semialdehyde → glycerate.

However, none of the above degradation products are likely to be found in significant concentrations in the environment due to their rapid rates of degradation. Pearce and Heydeman (1980) indicate that acetaldehyde, ethanol, and acetate were observed as aerobic degradation products of ethylene glycol.

Anaerobic degradation of ethylene glycol has been shown to occur by several authors (e.g., Dwyer and Tiedje, 1983), though the rate is typically slower than for aerobic degradation. Most authors suggest the pathway :

ethylene glycol → acetaldehyde → ethanol → acetate→ methane + CO_2.

Some or all of these compounds have been measured by various authors during anaerobic degradation.

The available literature (e.g., Sorensen et al., 1996) indicates that the most likely products of aerobic or anaerobic DEG or TEG degradation are acetaldehyde, ethanol, and acetate. The pathway is assumed to be hydrolysis of the ether linkages followed by degradation of ethylene glycol by the pathways discussed above.

3.5.2 Inhibition of Biodegradation

TEG was reported to have no inhibiting effects on biodegradation at 4,000 mg/L (Verschueren, 2001). No inhibition data were available for DEG or TREG.

3.5.3 Degradation Rate

The AENV (2009a) model for remediation guidelines protective of freshwater aquatic life includes a parameter value for the degradation rate of the chemical in an aquifer. The discussion of glycol degradation rates provided below is focussed on determining a suitable value for this parameter.

Data on the degradation rate of the Glycols are provided in Tables A-1, B-1, and C-1 for DEG, TEG, and TREG, respectively. Data in these tables are categorized based on whether the studies used amendments (activated sludge, additional carbon source, nutrients, electron acceptors, etc.) or were unamended.

Many datapoints are available in the above-noted tables for studies conducted under amended conditions ("Other Studies" in Tables A-1 and B-1). For example, Haines and Alexander (1975) investigated the biodegradation of EG, DEG, TEG, and TREG under aerobic conditions in a slurry of a silt loam and nutrient solution (including phosphate), amended with bacterial cultures acclimated to polyethylene glycol degradation. Based on an indirect method using biological oxygen demand, they found that all four compounds degraded readily and completely in 5 days or less under these favourable conditions. Thus, it appears that, with suitable amendments, all three glycols can degrade rapidly (a few days to a few weeks) and completely.

However, in practice, degradation rates for many compounds in groundwater are limited by the availability of nutrients and/or electron acceptors. Accordingly, the degradation rates for studies conducted under amended conditions may have little relevance to likely degradation rates in an aquifer, and are not discussed further in this document. Studies with data from unamended, or potentially relevant conditions are discussed below.

Kaplan et al. (1982) investigated the biodegradation of DEG and TEG under aerobic, anaerobic, and abiotic (sterile) conditions. They found that both compounds degraded at similar rates in abiotic conditions, as in aerobic or anaerobic reactors, with or without the addition of nutrients or glucose. They concluded that degradation of these compounds was primarily abiotic. Data from this study were interpreted to give abiotic half lives of 20 days for DEG and 35 days for TEG. It is noted that data from other studies do not support the relatively rapid abiotic degradation rates implied by Kaplan et al. (1982).

Mrklas et al. (2004) investigated the degradation of a mixture of monoethanolamine (MEA), EG, and TEG in slurries of contaminated soil and groundwater collected from a decommissioned sour gas plant. The study was designed with the objective of determining the potential for in-situ degradation of these compounds at the decommissioned sour gas plant. The initial level of TEG in the slurry was approximately 2,100 mg/kg. Aerobic and anaerobic studies were conducted on both biotic and abiotic bioreactors. TEG concentration was monitored directly using ion exclusion chromatography with pulsed amperometric detection. Biotic reactors received an addition of phosphate on day 11 or 64. Aerobic studies indicated that TEG degradation was limited by the availability of phosphate. Based on interpretation of data presented, in the absence of supplemental phosphate, the aerobic half life of TEG was approximately 175 days. With supplemental phosphate, the aerobic degradation of TEG was much more rapid, with a half life of approximately 25 days. Anaerobic data presented in the paper could not be interpreted to yield an anaerobic degradation rate.

Sorensen et al. (2000) conducted an extensive series of aerobic and anaerobic biodegradation tests of TEG and DEG with three soils, one each from oil-producing areas of Alberta, New Mexico, and Louisiana. All experiments were conducted at 25°C at 60% moisture-holding capacity in the dark, and degradation progress was monitored indirectly by CO_2 production (respirometry). Initial concentrations of 200 mg/kg and 1,000 mg/kg glycol were monitored using wet chemistry respirometry. Other concentrations were monitored using electronic respirometry. However, the electronic respirometry data were inconsistent with the wet chemistry results, and are not discussed here. For TEG, the time for half of the glycol to be degraded based on CO_2 production in aerobic biometers ("pseudo-half life") ranged from 11 days to 131 days under aerobic conditions. For DEG, the pseudo-half life based on aerobic tests at 200 mg/kg and 1,000 mg/kg glycol ranged from 16 days to 250 days. It is noted that most of the soil microcosms showed a lag time before significant biodegradation commenced. Lag times varied from 1-6 days for the more biologically active soils under aerobic conditions.

Sorensen et al. (2000) also conducted studies under anaerobic conditions, but these studies are not included in Tables A-1 or B-1. Respirometry is not an appropriate method to use for anaerobic studies since carbon dioxide can be lost under methanogenic conditions as it is a potential electron acceptor. Thus, the Sorensen et al. (2000) anaerobic studies are not discussed further.

TEG

Overall, the most relevant degradation study was considered to be Mrklas et al. (2004), based on the following considerations:

- **Unamended**. The study showed that TEG degradation can be phosphate limited, and the first 64 days of some tests were conducted without the addition of phosphate or other amendments.
- **Direct Analysis**. TEG degradation was monitored by direct chemical analysis, rather than an indirect method such as respirometry.
- **Relevant Substrate.** The study was conducted with a slurry of soil and groundwater from a decommissioned sour gas plant in Alberta that had used TEG.
- **Relevant Concentration.** Initial TEG concentrations were relevant to conditions at a sour gas plant in Alberta; and,
- **Relevant Moisture Content.** Data from this study on a slurry is more relevant to aquifer conditions than data from studies on soils at typical soil moisture contents.

The TEG half life of 175 days interpreted from the unamended parts of tests in the Mrklas et al. (2004) study has been selected for use in the calculation of remediation guidelines.

DEG

Mrklas et al. (2004) does not include data for DEG. The abiotic degradation rates implied in the Kaplan et al. (1982) study are considered suspect as they are not supported by other studies. The most relevant of the remaining data are the aerobic results from the Sorensen et al. (2000) study. This study uses a relevant substrate and concentrations (soils from three oilfield areas including Alberta), and is unamended. However, the study uses an indirect method (respirometry) to determine degradation and is conducted at a moisture content relevant to unsaturated soils rather than an aquifer. Based on the above considerations, this study is more relevant to determining an appropriate degradation rate for use in the groundwater transport model than other available data.

The DEG half life used in this study in the calculation of remediation guidelines is 250 days, based on the longest of the half lives interpreted from six aerobic tests (Sorensen, 2000) with three soils at two concentrations (Table A-1). It is noted that the two tests conducted with Alberta soils both had DEG half lives shorter than this.

3.6 Volatilization

All three glycols have negligible vapour pressure at room temperature, and hence volatilization will not have a significant effect on the transport and fate of glycols in the subsurface.

3.7 Photodegradation

A photodegradation half life of 11.4 hours was calculated for TEG (Verschueren, 2001). No relevant information was found on the photodegradation of either DEG or TREG.

4. BEHAVIOUR AND EFFECTS IN AQUATIC BIOTA

Toxicological data for freshwater aquatic life for the Glycols were compiled from the U.S. EPA ECOTOX database (U.S. EPA, 2007b) and other sources. The studies have undergone classification into Primary, Secondary, or Unacceptable/unverified categories with respect to the CCME (2006) protocol. Based on the CCME protocol, only Primary and Secondary data are used to develop water quality guidelines.

Data gaps in the minimum dataset required to develop at least interim freshwater aquatic life guidelines were identified and an additional study was commissioned from Vizon SciTec Inc. to fill the gaps (Vizon, 2006).

4.1 DEG

Freshwater aquatic toxicity data for DEG are provided in Table A-2. Eighteen datapoints of Primary or Secondary quality from seven studies are included (Figure 2). Five datapoints are for vertebrates, three are for invertebrates, three for plants (green algae), and seven for other biota.

The studies which include Primary and Secondary quality data are discussed in more detail in Section 10.2.1.

An additional two datapoints of Unacceptable data quality were available. These were classified as Unacceptable based on a lack of sufficient information to confirm that controls were acceptable. Fourteen other datapoints that did not show an effect at the highest concentration(s) tested are also included in Table A-2 for completeness, but are not considered in the guideline development process.

4.2 TEG

Freshwater aquatic toxicity data for TEG are provided in Table B-2. Fifty one datapoints of Primary or Secondary quality from seven studies are included (Figure 2). Twenty four datapoints are for vertebrates, twenty six are for invertebrates, and one for other biota.

The studies which include Primary and Secondary quality data are discussed in more detail in Section 10.2.2.

An additional two datapoints of Unacceptable data quality were available. These were classified as Unacceptable based on a lack of sufficient information to confirm that controls were acceptable. Twenty nine other datapoints that did not show an effect at the highest concentration(s) tested are also included in Table A-2 for completeness, but are not considered in the guideline development process.

4.3 TREG

No Primary or Secondary data were available for TREG, and only one Unacceptable/unverified data point was found. This data point is included in Table C-2 for completeness but are not discussed further.

4.4 Marine Biota

Toxicological data for marine aquatic life for DEG, TEG, and TREG are provided in Table A-3, Table B-3, and Table C-3, respectively. A total of 4, 27, and 1 marine data point(s) were identified for DEG, TEG, and TREG, respectively. The papers reporting these data have not been reviewed in detail. The studies have undergone preliminary classification into Primary, Secondary, or Unacceptable/unverified categories with respect to CCME protocol. However, it is possible that some data classified as Secondary or Unacceptable would be upgraded based on a review of the original paper.

Marine toxicity data are included in this literature search for completeness, but are not directly relevant to developing soil or groundwater quality guidelines in Alberta and are not discussed further.

5. BEHAVIOUR AND EFFECTS IN TERRESTRIAL BIOTA

5.1 Terrestrial Plants

No data were found in the literature on the toxicity of DEG, TEG, or TREG to terrestrial plants. Accordingly, definitive (14 or 21 day) growth tests were commissioned (Stantec, 2006) to assess the toxicity of DEG and TEG to three plant species, alfalfa (*Medicago sativa*), barley (*Hordeum vulgare*), and northern wheatgrass (*Elymus lanceolatus*). Environment Canada (2005a) toxicity test protocols were used for this work with minor modifications as documented in Stantec (2006). A full report on these tests is available at www.ptac.org, and the results are summarized in Tables A-4 (DEG) and B-4 (TEG). EC25 values for various endpoints for these three species ranged from 419 mg/kg to 2,742 mg/kg (DEG) and 1,924 mg/kg to 10,953 mg/kg (TEG). TEG was the less toxic of the two glycols tested. These data are analyzed in more detail in Section 12.1.

5.2 Soil Invertebrates

No data were found in the literature on the toxicity of DEG, TEG, or TREG to terrestrial invertebrates. Accordingly, chronic survival and reproduction tests were commissioned (Stantec, 2006) for two invertebrate species, the earthworm *Eisenia andrei*, and the springtail *Folsomia canadida*. Environment Canada (2004, 2005b) toxicity test protocols were used for this work with minor modifications as documented in Stantec (2006). A full report on these tests is available at www.ptac.org, and the results are summarized in Tables A-5 (DEG) and B-5 (TEG). EC25 values for reproduction endpoints for these two invertebrates ranged from 4,842 mg/kg to 7,697 mg/kg (DEG) and 7,528 mg/kg to 13,701 mg/kg (TEG). TEG was the less toxic of the two glycols tested. These data are analyzed in more detail in Section 12.1.

5.3 Soil Microbial Processes

No data were found on the toxicity or effects of DEG, TEG, or TREG to soil microbial processes.

6. BEHAVIOUR AND EFFECTS IN HUMANS AND MAMMALIAN SPECIES

Mammalian toxicological data for DEG, TEG, and TREG are provided in Tables A-6, B-6, and C-4, respectively. General aspects of the toxicology of these glycols are summarized below, and key studies are discussed. Effect and no-effect levels for selected mammalian toxicological studies on DEG and TEG are provided in Figure 3, where circles show human data, diamonds show acute animal data, and triangles show chronic animal data. Hollow symbols indicate no effect levels and solid symbols indicate effects. Only toxicological data for oral administration are plotted in Figure 3.

Soviet Studies

A part of the toxicological database for these compounds comprises Soviet studies, mostly from the 1980s. In many cases, it is difficult to reconcile the results of these studies with the wider body of global literature. Frequently the toxic endpoints reported are different from those reported in all non-Soviet studies, or in other cases, the effect concentrations reported are inconsistent with the other studies. Anecdotally, some of the Soviet work from this period is reputed to be of poor quality, and was not carried out to the same standards (*e.g.*, good laboratory practice standards; OECD, 1998) as are used in reputable studies. There is not typically sufficient information available to confirm the standards to which these studies were carried out. Accordingly, the results of Soviet studies are not included in the discussion that follows or in Figure 3.

6.1 Absorption, Biotransformation, and Elimination

In general, relatively little information on the absorption, biotransformation and elimination of glycols was found. However, one more detailed paper on these processes in TEG was available, and accordingly, that compound is reviewed in a little more detail than DEG or TREG in the following discussion.

DEG

Little information was found concerning the absorption, biotransformation and elimination of DEG. Absorption of DEG through the skin of rats is slow; only 10% of the applied neat dose (open, but protected application) being absorbed over a 72 hour exposure (Mathews et al., 1991). Absorption of DEG following oral exposure is assumed to be relatively rapid and complete, based on its physical and chemical properties, and by analogy with TEG, but no studies confirming this were found. Winek et al. (1978) state that the metabolic pathway of DEG is largely unknown, but they consider it unlikely that DEG is metabolized to formate, and thus the acidosis characteristic of acute ethylene glycol (and methanol) poisoning is not expected to be a prominent feature of DEG intoxication.

TEG

Triethylene glycol appears to be readily absorbed following oral exposure, with the majority of the compound being eliminated in urine (either as the parent compound, or as a metabolite) within a 5 day period.

A detailed study by McKennis et al. (1962) looked at the absorption and excretion of ^{14}C radio-labelled and unlabelled TEG in rabbits and rats. At the end of a 5 day period, the total recovery of ^{14}C from rats was 91-89% of the administered dose. The majority (93-97% of the recovered dose) was recovered from the urine with a small amount (2-6% of the recovered dose) in feces and only a trace (0.8-1.2%) in exhaled air. Much of the urinary excretion of radioactivity appeared during the first 24 hours following administration.

McKennis et al. (1962) also investigated the metabolic degradation of TEG by analysis of the urine produced. A significant part of the administered dose was recovered (by chloroform extraction) as unchanged TEG (26-34% in the first 24 hours in rabbits; 27-66% in rats). Subsequent acidification and re-extraction of one sample of rabbit urine residue yielded a further 35% of the administered dose. The metabolite extracted was not identified, but postulated to be ethylene glycol with one or both terminal hydroxyl groups oxidized to carboxylic acid. The authors subjected the urine to acid hydrolysis and chloroform extraction but concluded that essentially none of the TEG was excreted as either an ether or ester derivative. They also found essentially no ^{14}C activity in calcium oxalate precipitated from the urine samples, and concluded that the ether linkage was not broken during metabolism of TEG in rats. This is consistent with work by Schaffer et al. (1950) who studied the metabolism of polyethylene glycol 400 in man, and concluded that the metabolic cleavage of ether linkages to form ethylene glycol was not a significant process for this compound. The authors used the lack of ^{14}C in exhaled CO_2 to conclude that breaking of carbon-carbon linkages in TEG was not a significant metabolic pathway either. Lefaux (1968) confirmed that TEG is not metabolized to oxalic acid.

TREG

No information was found concerning the absorption, biotransformation, and elimination of TREG.

6.2 Acute Toxicity

DEG

The use of DEG in pharmaceuticals has caused the death of 71 adults in 3 separate incidents. Clinical findings included extensive kidney damage and less serious liver injury (BIBRA, 1993a). A similar range of acute symptoms has been reported in laboratory animals, where kidney and liver damage and central nervous system depression are the primary findings

(BIBRA, 1993a). Oral LD50 values for laboratory animals range from 3,300 to 32,000 mg/kg bw (Table A-6). Most studies found DEG to be non-irritant by dermal exposure.

TEG

Limited information from animal studies reveal a range of acute toxic effects overlapping those for DEG (BIBRA, 1993b). Smyth et al. (1941) found that rats and guinea pigs fed TEG at doses approaching the LD50 appeared sluggish (possibly indicating depression of the central nervous system) and gross examination revealed kidney damage. Oral LD50 values for laboratory animals range from 8,800 to 22,000 mg/kg bw (Table B-6). Ocular and dermal studies found TEG to be non-irritant or mildly irritating.

TREG

For oral exposure, published values of the LD50 ranged from 1,875-34,000 mg/kg bw (Table C-4). Target organs identified were liver and kidney (BIBRA, 1993c). An ocular study found TREG to cause only minimal irritation.

6.3 Sub-Chronic and Chronic Toxicity

DEG

Key animal toxicity studies on the sub-chronic and chronic toxicity of DEG are discussed below.

In an unpublished study (BIBRA, 1976), groups of 15 rats of each sex were fed a diet containing 0.085, 0.17, 0.4 or 2% DEG for 255 days or 4% for 99 days. Slight effects on kidney function and urine composition were seen at 0.4% (300 mg/kg bw/day). The only possible effect seen in rats on the 0.17% diet (100 mg/kg bw/day, Lowest Observable Effect Concentration [LOEC]) was a marginal increase in urinary oxalate in male rats. No effects were noted at 0.085% (50 mg/kg bw/day, No Observable Effect Concentration [NOEC]).

Freundt and Weis (1989) reported that female rats receiving DEG at 200 mg/kg bw/day for 90 days in their water showed no effect on kidney weight or urine biochemistry.

No overt toxic effects were seen in mice maintained for 15-18 weeks on a diet providing a DEG dose of about 5,200 mg/kg bw/day (Morrissey et al., 1988). However, Huber et al. (1986) exposed mice to DEG in their drinking water for 14-17 weeks, and reported effects on blood clotting and immune response at DEG doses as low as 50 mg/kg bw/day.

The results of these and other studies are summarized in Table A-6 and Figure 3.

TEG

Fitzhugh and Nelson (1946) exposed male rats to 4% TEG in their diet (approximately 2,000 mg/kg bw/day) for 2 years. No effects on mortality, body weight, blood and urine composition, and gross and microscopic appearance of the major organs was reported.

Robertson et al. (1947) exposed rats to TEG in their drinking water at 3,000 mg/kg bw/day for 13 months. No effects on mortality, body weight, blood and urine composition, and gross and microscopic appearance of the major organs was reported.

Bossert et al. (1992) exposed mice to drinking water containing TEG for 14 weeks. No effects were seen at 3,300 mg/kg bw/day, but increased liver weight was observed at 6,800 mg/kg bw/day.

The results of these and other studies are summarized in Table B-6 and Figure 3.

TREG

The chronic and sub-chronic dataset for TREG is poor. The results of two Soviet studies are summarized in Table C-4.

6.4 Carcinogenicity and Genetic Toxicity

DEG

Available carcinogenicity and genotoxicity information for DEG is summarized in Table A-6.

DEG is not thought to be a primary chemical carcinogen. Bladder tumours in male rats fed high levels of DEG for long periods were seen only in animals with bladder stones. The data of Weil et al. (1965, 1967) support the contention that the tumours resulted from the chronic irritation of the stones on the bladder wall. DEG at levels below levels known to induce stone formation would therefore be unlikely to pose a carcinogenic risk to animals.

In-vivo tests on hamsters via intraperitoneal injection produced slight chromosomal damage; oral exposure produced equivocal results. *In-vitro* tests produced no point mutation or chromosomal damage with mammalian cells, and there was no evidence of mutagenicity in Ames tests using the bacterium *Salmonella typhimurium*.

Overall, the available data do not support either carcinogenicity or genotoxicity being significant concerns for DEG.

TEG

Available carcinogenicity and genotoxicity information for DEG is summarized in Table B-6.

No evidence of carcinogenicity was found in a 2 year study in which groups of 12 male rats received diets containing TEG at up to 2,000 mg/kg bw/day. Microscopic examinations were made of tissues from the major organs (Fitzhugh and Nelson, 1946).

The genotoxicity database for TEG is limited. Apart from some Soviet studies indicating various positive results, the only other information is an indication that TEG was genotoxic in an Ames bacterial test (no further details available, NTP, 1991).

Overall, the available data do not support either carcinogenicity or genotoxicity being significant concerns for TEG.

TREG

No human or animal data were found that were relevant in determining the status of TREG as a carcinogen.

Available genotoxicity information for TREG is summarized in Table C-4. No evidence of mutagenicity was seen in an Ames tests using the bacterium *Salmonella typhimurium*, or in mammalian cells in culture. There was an increase in chromosomal effects (sister chromatid exchange and damage) in mammalian cells, but the effect was weak and there was no dose-related trend. In mice given 5,000 mg/kg bw by intraperitoneal injection, chromosomal damage was induced in the peripheral blood cells, but rats given 5,000 mg/kg bw orally did not exhibit any increase of chromosomal damage in the bone marrow cells (BIBRA, 1993c).

Overall, the weight of available evidence does not suggest that genotoxicity is a significant concern for TREG. No direct evidence is available to assess the potential for carcinogenicity, however, analogy with DEG and TEG does not suggest that carcinogenicity is likely to be a significant concern for TREG.

6.5 Reproduction and Developmental Toxicity

DEG

Studies on the reproductive and developmental toxicity of DEG are summarized in Table A-6. The key studies are discussed briefly below.

The data for DEG indicate that developmental and reproductive endpoints are less sensitive than the effects seen in the chronic BIBRA (1976) study (NOEC = 50 mg/kg bw/day). Rodwell et al. (1987) found no effect on reproduction at 500 mg/kg bw/day when rats were exposed to DEG in drinking water continuously for 2 generations. Rodwell et al. (1987) found increased kidney weights in the parental and first generations at 1,500 mg/kg bw/day.

TEG

Studies on the reproductive and developmental toxicity of TEG are summarized in Table A-6. The key studies are discussed briefly below.

In a continuous breeding study, Bossert et al. (1992) exposed mice to TEG. Slightly reduced pup weight was noted at 3,400 mg/kg bw/day (LOEC), but no impairment of reproductive efficiency was noted in the first generation at 80 days. No effect on pup weight was noted at 680 mg/kg bw/day (NOEC).

Similar results were reported by U.S. EPA (1990), who found a NOEC of 600 mg/kg bw/day and a LOEC of 5,600 mg/kg bw/day for reduced fetal weight, reduced ossification, and increased skeletal variations when mice were exposed to TEG on day 6-15 of pregnancy.

Rats may be less sensitive than mice to reproductive and developmental effects of TEG, based on a 13 month continuous breeding study by Robertson et al. (1947), who found no overt effect on reproduction at 3,000 mg/kg bw/day.

TREG

There is little confidence in the quality of the small amount of available information on the reproductive toxicity of TREG (Table C-4).

6.6 Tolerable Daily Intake

Tolerable daily intake (TDI) is the daily oral dose of a contaminant that is assumed to be sufficiently low that humans could be exposed at this dose over an entire lifetime without adverse effects. The tolerable daily intakes identified for the Glycols are summarized below.

DEG

The European Community (EC)'s Scientific Committee on Food assigned a human TDI of 0.5 mg/kg bw/day for the intake of DEG (European Commission, 2008). This TDI is consistent with applying an uncertainty factor of 100 to the NOEC from the chronic BIBRA (1976) study, and is the value used in this document (Table 3).

TEG

The EC's Scientific Committee for Food (European Commission, 2003) assigned a TDI for man of 5 mg/kg bw/day for the combined intake of TEG and polyethylene glycol (European Commission, 2003). This TDI is consistent with applying an uncertainty factor of 100 to the NOEC from the Bossert et al. (1992) reproduction study, and rounding down, and is the value used in this document (Table 3).

TREG

The EC's Scientific Committee for Food assigned a TDI for man of 10 mg/kg bw/day for TREG (European Commission, 2003).

7. TOXICITY OF DEGRADATION PRODUCTS

In certain cases, organic compounds can have degradation products that are more toxic than the parent compound. Prudent management of such a parent compound should take into consideration the possibility of more toxic degradation products. A complete review of the toxicity of degradation products is outside the scope of the current study. However, it is worth noting that Sorensen et al. (1996) highlight formaldehyde, acetaldehyde, and ethanol as being potential glycol degradation products that are, or may be, more toxic than the parent compounds. Both formaldehyde and acetaldehyde are considered to be more toxic than DEG, with formaldehyde being the most toxic of these compounds. The Dutch environmental regulators (VROM, 2000), provide "indicative levels for serious contamination" for formaldehyde in soil and groundwater of 0.1 mg/kg and 0.05 mg/L, respectively. These values are 2-3 orders of magnitude lower than the corresponding values for DEG.

8. DATA ADEQUACY AND DATA GAPS

The available data were assessed against AENV (2009a) and CCME (2006) requirements for developing soil and water quality guidelines.

8.1 Soil Quality Guidelines

Human Health Guidelines

Sufficient data are available to develop soil quality guidelines protective of human soil ingestion, potable groundwater, and off-site migration. The indoor air inhalation guideline is not required, since the Glycols are not volatile. The guideline protective of ingestion of produce, milk and meat is not required, since the Glycols are not expected to biomagnify, based on their K_{ow} values.

Ecological Guidelines

None of the data available in the literature were suitable for calculating a soil contact guideline. A study was commissioned, which filled this data gap (Stantec, 2006).

None of the available data are suitable for calculating the nutrient and energy cycling check. A soil quality guideline can be calculated without this check.

Sufficient information was available to calculate a soil quality guideline protective of freshwater aquatic life, based on the surface water quality guideline for freshwater aquatic life discussed in Section 8.2.

Insufficient data exist to calculate the soil and food ingestion guideline. The CCME (2006) protocol for this guideline requires toxicity data from tests conducted on livestock species, and these data do not currently exist.

8.2 Water Quality Guidelines

Drinking Water

Sufficient data are available to develop Source Guidance Values for Groundwater based on the tolerable daily intake values discussed in Section 6.6.

Freshwater Aquatic Life

A study was commissioned (Vizon, 2006) to fill gaps in the freshwater aquatic life dataset. Including the new data from this study, there is sufficient information to develop interim freshwater aquatic life water quality guidelines for DEG and TEG. Insufficient data exist to develop a freshwater aquatic life water quality guideline for TREG.

Irrigation Water

Available data are currently insufficient to calculate an irrigation water guideline for any of the Glycols. In order to meet the CCME (1993) requirements to calculate this guideline for DEG and TEG, two additional toxicological studies would be required for each chemical, one on a cereal, tame hay, or pasture crop, and one on another crop.

Livestock Watering

Insufficient data are available to meet the requirements published in CCME (1993) for developing a livestock watering guideline.

9. PARAMETER VALUES

Parameter values required to calculate Alberta Tier 1 soil and groundwater remediation guidelines for DEG and TEG fall into two main groups: i) parameters that relate to the chemical properties, toxicity, or background exposure to the Glycols, referred to as "chemical-specific parameters"; and, ii) parameters relating to receptor exposure and properties of the site, referred to as "non-chemical-specific parameters". These two groups of parameters are discussed below.

9.1 Chemical-Specific Parameters

Chemical-specific parameters for DEG and TEG are summarized in Table 3, together with an indication of where to find a discussion of the rationale for the value selected. The soil allocation factor (SAF) and water allocation factor (WF) each take the values of 0.25, since exposure to DEG and TEG could reasonably be anticipated via four potentially contaminated environmental media: soil, water, food, and consumer products. However, exposure via air, the fifth potentially-contaminated medium, is unlikely due to the negligible vapour pressure of the Glycols (Section 2.6).

9.2 Non Chemical-Specific Parameters

Non chemical-specific parameter values are taken without change from AENV (2009a). Parameter values for human receptor characteristics, soil and hydrogeological parameters, site characteristics, and building parameters are provided in Tables 4 to 7, respectively.

10. SURFACE WATER GUIDELINES

AENV and the CCME use surface water quality guidelines as a basis from which to calculate corresponding groundwater and soil quality guidelines. Surface water quality guidelines calculated for DEG and TEG are provided and discussed below.

10.1 Human Drinking Water

No Canadian Drinking Water Quality Guideline (CDWQG) currently exists for any of the Glycols. In such cases, CCME (2006) includes a protocol for calculating an allowable concentration in potable water (Source Guidance Value for Groundwater) from the tolerable daily intake using the following equation:

$$SGVG = \frac{TDI \times BW \times WF}{WIR}$$

where:

SGVG = Source Guidance Value for Groundwater (mg/L)
TDI = tolerable daily intake (mg/kg/d)
BW = body weight (kg)
WF = water allocation factor (unitless)
WIR = water ingestion rate (L/d)

The SGVG is calculated using adult parameters (CCME, 2006). Substituting appropriate parameter values from Tables 3 and 4 gives values of 5.9 mg/L (DEG) and 59 mg/L (TEG). These values are rounded to 1 significant figure with a 5 or 0 in the second figure to give 6 mg/L (DEG) and 60 mg/L (TEG) which are the Source Guidance Values for Groundwater for these compounds (Table 8).

10.2 Freshwater Aquatic Life

Interim freshwater aquatic life water quality guidelines for DEG and TEG were calculated based on the CCME (1991) protocol. Freshwater aquatic toxicity data were obtained from the U.S. EPA ECOTOX database and other sources discussed in Section 4, and are summarized in Tables A-2 and B-2, for DEG and TEG, respectively.

Data Quantity Requirements

Insufficient data exist for the development of full freshwater aquatic life water quality guidelines for DEG or TEG. However, minimum data requirements are met for both chemicals for the development of an interim guideline (two acute and/or chronic studies on two or more fish species, including one cold water species resident in North America; two acute and/or chronic studies on two or more invertebrate species from different classes, including one planktonic species). Thus, it was possible to develop interim freshwater aquatic life water quality guidelines for DEG and TEG.

Data Quality Screening

Wherever possible, all identified studies were identified and reviewed. In some cases, (e.g., foreign language journals and databases, data in books that are out of print) the original source could not be obtained, and it was necessary to rely on the ECOTOX reviewers for key study elements such as endpoints and acceptability of controls. Datapoints were assigned to Primary, Secondary, or Unacceptable categories, based on the CCME (1991) criteria. The most common reason for a study being categorized as Unacceptable was a lack of information indicating that controls were conducted and that control response was acceptable. Data where the concentration reported in the ECOTOX database was ">x" are included for completeness in a separate category in Tables A-2 and B-2. These data contain no information on concentrations at which effects are seen, and are not considered in the guideline development process. Where the ECOTOX database reported the same value for the same species and same author, this information is assumed to be redundant, and is only presented once in Tables A-2 and B-2.

Ecological Relevance

Guidelines are developed from ecologically relevant data. Accordingly, the toxicity endpoints in Tables A-2 and B-2 were screened for relevance to the ecological health of freshwater aquatic ecosystems.

10.2.1 DEG

The Primary and Secondary data for DEG included seven studies, details are summarized in Table A-2, and discussed below.

Bringmann and Kuhn (1980). This paper summarized the results of tests on 156 industrial pollutant chemicals on a bacterium (*Pseudomonas putida*), a green alga (*Scenedesmus quadricauda*), and a protozoan (*Entosyphon sulcatum*). All three species have been identified as being involved in the bioremediation of pollutant chemicals. These tests were evaluated to estimate the concentration of contaminant that would result in a 3% reduction in growth relative to controls (IC03) over the time period specified. The most sensitive of these species to DEG was *Scendesmus quadricauda*), with a 7 day IC03 of 2,700 mg/L. This test duration was considered chronic relative to typical unicellular algae cell proliferation rates.

de Zwart and Sloof (1987). This study was designed to investigate the toxicity of mixtures of chemicals, but also includes 48 hour LC50 values for 3-4 week old clawed toad larvae (*Xenopus* *laevis*) exposed to 33 single chemicals including DEG. The 48 hour (acute) LC50 for this species for DEG was 3,065 mg/L.

Geiger et al. (1990). This book is a large compilation of acute toxicity data for the Fathead minnow, and is out of print. The Fathead minnow LC50 for DEG from the ECOTOX database reported in Table A-2 (75,200 mg/L) is broadly consistent with the Vizon (2006) LC50 for rainbow trout, and the Geiger et al. (1990) data are not limiting in the development of a DEG guideline. Accordingly, the original source was not reviewed for this datapoint.

Ward et al. (1992). A copy of this unpublished study was kindly provided for review by Environment Canada. Acute mortality studies were conducted on two freshwater fish (fathead minnow – *Pimephales promelas* and rainbow trout – *Oncorhynchus mykiss*), and one freshwater invertebrate (*Daphnia magna*). A chronic growth test was performed on the green alga *Selanastrum capricornutum*. Tests were also conducted on three marine species (Table A-3). Results for the animal species were consistent with other studies. Growth of *S. capricornutum* was measured at a range of time periods from 24 hours to 14 days, and appeared to indicate higher toxicity at earlier times. One possible reason for this could be the alga becoming acclimated to the toxicant. The concentration of DEG was relatively stable throughout the 14 day test, with the average measured concentration at 14 days being 79% of nominal. The 14 day result was considered to be the most relevant to the long-term health of an aquatic ecosystem, and accordingly the 14 day IC50 and LOEC are included in Table A-2.

Sauvant et al. (1995a,b). These two studies investigated the toxicity of a range of chemicals to *Tetrahymena pyriformis*. *T. pyriformis* is a ciliated protozoan (single-celled organism) found in freshwater bodies around the world. Accordingly, it is ecologically relevant to developing freshwater aquatic life water quality guidelines. The lowest reported LC50 value for the growth of this organism was 22,500 mg/L for a 36 hour test. All results in these studies are considered chronic, since the durations of the tests were long, compared with the doubling rate of 3 h for these protozoa.

Vizon (2006). This study is available at www.ptac.org and was commissioned to fill data gaps in the literature such that at least the minimum requirements for developing a CCME interim guideline were met. Vizon (2006) conducted 96 hour static lethality tests using rainbow trout and the freshwater amphipod *Hyalella azteca,* and a 48 hour static lethality test using *Daphnia magna*. Environment Canada biological test methods were used throughout (EPS 1/RM/9 for rainbow trout, EPS 1/RM/33 for *Hyalella azteca*, and EPS 1/RM/11 for Daphnia magna). All the requirements for Primary data quality were met, including measured chemical concentrations. Results are provided in Table A-2. The lowest acute LC50 was 63,000 mg/L, which was the 48 hour result for *D. magna*.

The CCME (1991) protocol for calculating the guideline considers Primary and Secondary data and takes the lower of:

1. the lowest LOEC for a chronic study for a non-lethal endpoint is multiplied by a safety factor of 0.1.
2. The lowest EC50 or LC50 for an acute test is multiplied by an application factor of 0.05 (DEG is considered non-persistent in surface water based on the degradation data provided in Table A-1 for conditions where oxygen and nutrients are not limiting).

Chronic Studies

The lowest endpoint from a chronic study among the Primary and Secondary data in Table A-2 is 2,700 mg/L, which is the Bringmann and Kuhn (1980) IC03 for growth inhibition in the green alga *Scenedesmus quadricauda.* Therefore, a freshwater aquatic life water quality guideline based on a chronic study is calculated by multiplying the IC03 of 2,700 mg/L from this study by a safety factor of 0.1 to give a guideline value of 270 mg/L.

Acute Studies

The freshwater guideline derived from the lowest relevant acute EC50/LC50 is calculated by multiplying the de Zwart and Sloof (1987) 48 hour LC50 for the clawed toad *Xenopus laevis* (3,065 mg/L) by an application factor of 0.05 (non-persistent variable) to give a guideline value of 153 mg/L.

The guideline value from the acute study is the lower of the two values calculated above, and accordingly, the freshwater aquatic life water quality guideline for DEG is 153 mg/L. This value is rounded to 1 significant figure with a 5 or 0 in the second figure to give 150 mg/L (Table 8).

10.2.2 TEG

Many of the studies on the freshwater aquatic toxicity of TEG were conducted to assess the potential effects of TEG when used as a solvent in toxicity tests of lipophilic chemicals ("carrier solvent"). The Primary and Secondary data in Table B-2 include the following eight studies, discussed below.

Cardwell et al. (1978). A copy of this unpublished study was kindly provided for review by the Mid-Continent Ecology Division of the U.S. EPA in Duluth, Minnesota. This detailed report provided information on a study that was undertaken to investigate the acute and chronic toxicity of TEG and 3 other carrier solvents to fathead minnow (*Pimephales promelas*), brook trout (*Salvelinus fontinalis* Mitchill), and bluegill (*Lepomis macrochirus* Rafinesque). The summary data on this study included in the U.S. EPA (2007b) ECOTOX report contain some inaccuracies relative to the content of the report, and Table B-2 reflects the source report rather than the ECOTOX summary.

- ***Acute Tests***. Acute lethality tests were conducted in flow-though aquaria for all three species with test durations ranging from 12 h to 7 days (168 hours). Static acute tests were also conducted with fathead minnows only. Results are summarized in Table B-2. The most sensitive species was the bluegill, and the lowest acute LC50 was the 7 day result for bluegill, 60,157 mg/L.

- ***Chronic Tests***. Significant resources were expended in conducting two generation chronic tests with brook trout (15 months total duration) and fathead minnow (12 months total duration). A wide range of endpoints were considered in each test, including hatching success, mortality, length, weight and spawning success of the F_0 generation, and hatching success, mortality, length, and weight of the F_1 generation. No significant effect was seen on the growth (length or weight) of either the F_0 or the F_1 generations. Unfortunately, utility of the results from these tests was compromised by the following factors:
    - The study design did not have adequate power to determine statistical significance for many of the endpoints.
    - Maximum test concentrations were not high enough to determine effect levels for many of the endpoints considered.
    - On two instances, inadvertent chlorination of the supply water caused significant mortality of the brook trout alevins, and compromised the data from that test.
    - Some fish were damaged by handling and/or fighting during the tests, and developed infections of the fungus *Saprolegnia parasitica*. Either the fungus or the attempted treatment proved fatal to these fish.
    - Several fish developed apparent bacterial hemorrhagic septicemia, which was treated by the antibiotic oxytetracycline, adding further uncertainty to the test results.
    - All aquaria became contaminated with a bacterium believed to be *Sphaerotilus* sp. which proliferated to such an extent that twice weekly cleaning of aquaria was required. In the higher concentration treatments, bacterial growth was observed on the surface of the eggs, which was postulated by the authors to reduce the availability of oxygen to the eggs and potentially be a cause of the increased number of abnormal fry at hatch seen in some treatments.

Overall, the data from the Cardwell et al. (1978) chronic tests were considered to be irrevocably compromised by the factors noted above, and the data are not included in Table B-2 or considered further in the development of a water quality guideline.

Bringmann and Kuhn (1978). This study was not obtained for review (foreign language journal), however, based on the data reported in the ECOTOX database and extrapolation from other similar work conducted by these authors, it is likely that acceptable controls were included. The study investigated the toxicity of TEG to the blue-green alga *Microcystis aeruginosa*. Since it appears that algae are some of the most sensitive organisms to glycols, the precautionary principle dictates that the results of this study should be taken at face value. Two other studies by the same authors reported the same value for another species of blue-green alga. The study reported a LOEC for 8 day *Microcystis aeruginosa* growth of 3,600 mg/L. This duration is considered chronic for algae.

Barera and Adams (1983). This study examined various aspects of the American Society for Testing and Materials (ASTM) standard for conducting acute toxicity tests with Daphnia magna, including the use of carrier solvents such as TEG. Based on the information provided in the paper, the study appears to be of high quality in all respects including the reporting of controls. Chemical concentrations were nominal, rather than measured, and accordingly, the study is designated of Secondary data quality. The study reports a 24 hour LC50 of 88,500 mg/L, a 48 hour LC50 of 52,400 mg/L, and a 48 hour mortality NOEC of 24,000 mg/L.

LeBlanc and Surprenant (1983). This study was designed to validate the use of 3 carrier solvents including TEG in toxicity tests with *Daphnia magna*. Based on the information provided in the paper, the study appears to be of high quality in all respects including the reporting of controls. Chemical concentrations were nominal, rather than measured, and accordingly, the study is designated of Secondary data quality. Acute mortality tests were conducted in static test vessels, and yielded a 24 hour LC50 of 58,000 uL/L (65,250 mg/L) and a 48 hour LC50 of 35,000 uL/L (39,375 mg/L). Chronic 28 day survival and reproduction tests were conducted in flow-through aquaria, and a LOEC of 11,000 uL/L (12,375 mg/L) was determined for both survival and reproduction.

Adams and Heidolph (1985). This study was designed to develop application factors used to extrapolate the results of *Daphnia magna* acute or partial life-cycle tests to the anticipated result for a 21 day geometric mean maximum acceptable toxicant concentration (GM-MATC). This large study examined eight test chemicals including TEG. Based on the information provided in the paper, the study appears to be of high quality in all respects including the reporting of controls. Chemical concentrations were nominal, rather than measured, and accordingly, the study is designated of Secondary data quality. Acute mortality tests (24 and 48 hour) were conducted in static test vessels. Results from acute tests appear to be the same data as reported by Barera and Adams (1983). Chronic tests considered growth, survival and reproduction at 7, 14, and 21 days and were conducted under renewal conditions. Results are provided in Table B-2. The lowest acute LC50 in this study was 42,426 mg/L (measured at day 2 of the chronic test). The lowest chronic LOEC was 15,000 mg/L for *D. magna* growth at 21 days.

Ziegenfuss et al. (1986). This study was designed to investigate the effect of water-sediment partitioning on the toxicity of a range of chemicals, including TEG, on one benthic invertebrate (*Chironomus tentans*) and one free-swimming invertebrate (*Daphnia magna*). Toxicity data were also included for standard acute toxicity tests (without sediment).

Geiger et al. (1990). This book is a large compilation of acute toxicity data for the fathead minnow, and is out of print. The data for TEG from the ECOTOX database reported in Table B-2 are broadly consistent with the other fish toxicity data for this chemical, and the original source for these data was not reviewed.

Vizon (2006). This study is available at www.ptac.org and was commissioned to fill data gaps in the literature such that at least the minimum requirements for developing a CCME interim guideline were met. Vizon (2006) conducted a 96 hour static lethality test using the freshwater amphipod *Hyalella azteca*. Environment Canada biological test method EPS 1/RM/33 was used and all the requirements for Primary data quality were met, including measured chemical concentrations. The 48 hour LC50 was 43,500 mg/L, essentially consistent with the data for *D. magna* determined in other studies.

The CCME (1991) protocol for calculating the guideline considers Primary and Secondary data and takes the lower of:

1. the lowest LOEC for a chronic study for a non-lethal endpoint is multiplied by a safety factor of 0.1.
2. The lowest EC50 or LC50 for an acute test is multiplied by an application factor of 0.05 (TEG is considered non-persistent in surface water based on the degradation data provided in Table B-1 for conditions where oxygen and nutrients are not limiting).

Chronic Studies

The lowest endpoint from a chronic study among the Primary and Secondary data in Table B-2 is 3,600 mg/L which is the 8 day LOEC for *Microcystis aeruginosa* growth in the Bringmann and Kuhn (1978) study. Therefore, a freshwater aquatic life water quality guideline based on a chronic study is calculated by multiplying the LOEC of 3,600 mg/L from this study by a safety factor of 0.1 to give a guideline value of 360 mg/L.

Acute Studies

The lowest endpoint from an acute study among the Primary and Secondary data in Table B-2 is 39,375 mg/L which is the 48 hour LC50 for *Daphnia magna* mortality in the LeBlanc and Surprenant (1983) study. The freshwater guideline derived from the lowest relevant acute LC50 is calculated by multiplying this value by an application factor of 0.05 (non-persistent variable) to give a guideline value of 1,969 mg/L.

The guideline from the chronic study is the lower of the two guidelines calculated, and accordingly, the freshwater aquatic life water quality guideline for TEG is 360 mg/L. This value is rounded to 1 significant figure with a 5 or 0 in the second figure to give 350 mg/L (Table 8).

10.3 Irrigation Water

No guideline was calculated for the Glycols in irrigation water since the minimum data requirements were not met (Section 8.2).

10.4 Livestock and Wildlife Watering

Toxicity data for the Glycols were not available for livestock or wildlife species (Section 8.2), and accordingly, these guidelines could not be calculated.

11. SOIL AND GROUNDWATER GUIDELINE CALCULATIONS – HUMAN HEALTH

11.1 Direct Contact

The model used to calculate the soil quality guideline protective of the human direct soil contact (soil ingestion, dermal contact, and particulate inhalation) exposure pathway for the Glycols is taken without change from AENV (2009a). Parameter values are summarized in Tables 3 and 4. The following equation was used.

$$PSQG_{HH} = \frac{(TDI - EDI) \times SAF \times BW}{\left[(AF_G \times SIR) + (AF_L \times IR_S \times ET_2) + (AF_S \times SR)\right] \times ET_1} + [BSC]$$

Where:

$PSQG_{HH}$ = preliminary human health-based soil quality guideline (mg/kg)
TDI = tolerable daily intake (mg/kg bw per day)
EDI = estimated daily intake (mg/kg bw per day)
SAF = soil allocation factor (dimensionless)
BW = adult or toddler body weight (kg)
AF_G = absorption factor for gut (dimensionless)
AF_L = absorption factor for lung (dimensionless)
AF_S = absorption factor for skin (dimensionless)
SIR = adult or toddler soil ingestion rate (kg/day)
IR_S = inhalation of particulate matter re-suspended from soil (kg/day)
SR = adult or toddler soil dermal contact rate, see below (kg/day)
ET_1 = exposure term 1 (dimensionless) (days/week ÷ 7 x weeks/year ÷ 52)
ET_2 = exposure term 2 (dimensionless) (hours/day ÷ 24)
BSC = background soil concentration (mg/kg)

Substituting appropriate values from Tables 3 and 4 into this equation and rounding to 1 significant figure with a 5 or 0 in the second figure gives human direct contact guideline values of:

DEG (Tables 9 and 10):

- 15,000 mg/kg (agricultural and residential);
- 20,000 mg/kg (commercial); and,
- 100,000 mg/kg (industrial).

TEG (Tables 11 and 12):

- 150,000 mg/kg (agricultural and residential); and,
- 200,000 mg/kg (commercial).

- no guideline is required ("ngr" in Tables 11 and 12) for industrial land use since the calculated value is $>10^6$ mg/kg.

Soil Dermal Contact Rate

The soil dermal contact rate (SR) is the mass of contaminated soil which is assumed to contact the skin each day. This parameter is calculated as follows (AENV, 2009a):

$$SR = \{(SA_H \times DL_H) + (SA_O \times DL_O)\} \times EF$$

Where:

SR = soil dermal contact rate (kg/day)
SA_H = exposed surface area of hands (m^2)
DL_H = dermal loading of soil to hands (kg/m^2 per event)
SA_O = area of exposed body surfaces other than hands (m^2)
DL_O = dermal loading of soil to other surfaces (kg/m^2 per event)
EF = exposure frequency (events/day)

The soil dermal contact rate is calculated separately for toddlers and adults using the parameters in Table 4, and is 6.88×10^{-5} kg/day for toddlers, and 1.14×10^{-4} kg/day for adults.

11.2 Inhalation

The Glycols are effectively non-volatile (Section 3.6) and accordingly remediation guidelines protective of the indoor air inhalation exposure pathway are not calculated for either soil or groundwater.

11.3 Offsite Migration

Offsite Migration guidelines are calculated to check that the guidelines set for commercial and industrial land use will not result in adjacent more sensitive land being contaminated at levels above the applicable guideline for the sensitive land due to wind and/or water transport of contaminated soil from the commercial or industrial site. The human health offsite migration guideline is calculated using the equation provided in AENV (2009a):

$$SQG_{OM} = (14.3 \times SQG_A) - (13.3 \times BSC)$$

Where SQG_{OM}= soil quality guideline protective of offsite migration (mg/kg)
SQG_A = soil quality guideline for human direct soil contact for agricultural land use (mg/kg)

BSC = background soil concentration (mg/kg)

Substituting appropriate values from Tables 3, 9, 10, 11, and 12 into this equation and rounding to 1 significant figure with a 5 or 0 in the second figure gives a human health offsite migration guideline of 200,000 mg/kg for DEG; Tables 9 and 10. No guideline is required ("ngr" in Tables 11 and 12) for TEG since the calculated value is $>10^6$ mg/kg.

12. SOIL AND GROUNDWATER GUIDELINE CALCULATIONS – ECOLOGICAL

12.1 Direct Contact

12.1.1 Soil

The soil quality guideline for the exposure pathway considering direct contact of plants and soil invertebrates (the "eco-contact pathway") was calculated for DEG and TEG based on a weight of evidence approach following CCME (2006). Data relevant for guideline development are sourced from Stantec (2006) (available at www.ptac.org) and are summarized in Tables A-4 and A-5 (DEG) and B-4 and B-5 (TEG). The values provided in the above-noted tables are nominal values based on the known amount of chemical spiked into the test soils. Stantec (2006) included analytical data to confirm exposure concentrations. Analytical data from day 0 in the definitive tests were analyzed to give the following regressions:

$$y = 1.0184x - 220.28 \text{ (DEG)}$$

$$y = 1.0145x - 256.93 \text{ (TEG)}$$

where x is the nominal concentration and y the measured concentration.

		IC25 (Corrected for Analytical Recovery)	
Species	**Effect**	**DEG (mg/kg)**	**TEG (mg/kg)**
Alfalfa	Shoot Length	1,101	7,005
Alfalfa	Root Length	1,296	9,543
Alfalfa	Shoot Dry Mass	2,359	6,454
Alfalfa	Root Dry Mass	2,297	7,284
Barley	Shoot Length	2,536	7,530
Barley	Root Length	2,572	10,855
Barley	Shoot Dry Mass	206	4,120
Barley	Root Dry Mass	766	4,949
Northern Wheatgrass	Shoot Length	1,552	4,887
Northern Wheatgrass	Root Length	1,703	5,533
Northern Wheatgrass	Shoot Dry Mass	613	1,695
Northern Wheatgrass	Root Dry Mass	919	1,915
Eisenia andrei	Number of Progeny	7,618	9,298
Eisenia andrei	Dry Mass of Individual Progeny	4,711	7,380
Folsomia candida	Number of Progeny	5,219	13,643

These regressions indicate essentially quantitative recovery of these glycols from the test soils. The CCME (2006) protocol uses data standardized at the 25th percentile effect level. Invertebrate survival data were not calculated at the 25% effect level by Stantec (2006), and were not included in the calculation of guideline values. The data that were used to calculate the eco-contact guideline are presented below. These data have been corrected for analytical recovery from the values in Tables A-4, A-5, B-4, and B-5.

The 25th percentile of these data is the eco-contact guideline for natural areas, agricultural, and residential. The 50th percentile of these data is the eco-contact guideline for commercial and industrial land use. The eco-contact guidelines for DEG and TEG are summarized below (rounded to 1 significant figure with a 5 or a 0 as the second figure) and included in Tables 9, 10, 11, and 12.

DEG

- 25th percentile - natural areas, agricultural, and residential: 1,000 mg/kg.
- 50th percentile - commercial and industrial: 1,500 mg/kg.

TEG

- 25th percentile - natural areas, agricultural, and residential: 5,000 mg/kg.
- 50th percentile - commercial and industrial: 7,000 mg/kg.

12.1.2 Groundwater

The direct contact of shallow groundwater with plants and soil invertebrates exposure pathway is applicable whenever groundwater is present within 3 m of the ground surface. However, based on guidance in AENV (2009a), the guideline is not calculated for polar compounds such as the Glycols. The rationale for this position is that the potential interactions between polar organic compounds and soils are complex in that they can be highly dependant on various environmental conditions including pH, clay mineralogy, and redox conditions. Attempting to set groundwater guidelines for polar chemicals for this pathway would involve significant uncertainty, and accordingly, it is recommended that concerns with potential adverse effects on surface soil biota from polar organic compounds in shallow groundwater be addressed on a site-specific basis by analyzing soil samples.

Accordingly, the groundwater guideline protective of the eco-contact pathway is not calculated for the Glycols.

12.2 Nutrient and Energy Cycling

Insufficient data were available and this guideline was not calculated for the Glycols.

12.3 Soil and Food Ingestion

Insufficient data were available (Section 8.1), and this guideline was not calculated for the Glycols. However, this exposure pathway was not expected to be a concern, since i) the Glycols are expected to degrade rapidly in surficial soil (Section 3.5) and accordingly livestock and wildlife are unlikely to get significant exposure to the Glycols through incidental ingestion of surficial soil; and ii) based on their very low K_{ow} values (negative log K_{ow}; Table 2) DEG and TEG are not expected to accumulate into plants to any significant extent; thus, the exposure of livestock or wildlife to DEG and TEG in soil via ingestion of fodder is expected to be minimal.

12.4 Offsite Migration

Offsite Migration guidelines are calculated to check that the guidelines set for commercial and industrial land use will not result in adjacent more sensitive land being contaminated at levels above the applicable guideline for the sensitive land due to wind and/or water transport of contaminated soil from the commercial or industrial site. The ecological offsite migration guideline is calculated using the equation provided in AENV (2009a):

$$SQG_{OM} = (14.3 \times SQG_A) - (13.3 \times BSC)$$

Where

SQG_{OM}= soil quality guideline protective of offsite migration (mg/kg)

SQG_A = soil quality guideline for ecological direct soil contact for agricultural land use (mg/kg)

BSC = background soil concentration (mg/kg)

Substituting appropriate values from Tables 3, 9, 10, 11, and 12 into this equation and rounding to 1 significant figure with a 5 or 0 in the second figure gives ecological offsite migration guidelines of 15,000 mg/kg for DEG (Tables 9 and 10), and 70,000 mg/kg for TEG (Tables 11 and 12).

13. SOIL AND GROUNDWATER GUIDELINE CALCULATIONS – GROUNDWATER PATHWAYS

This section provides the protocols used to calculate soil and groundwater remediation objectives protective of exposure pathways involving groundwater. The following receptors are considered:

- humans (potable drinking water sourced from groundwater); and,
- aquatic life (via lateral groundwater transport and discharge into a surface water body).

In the first case, it is assumed that a water well could potentially be installed at any location, and hence it is assumed that there is no lateral offset between the location where the contaminated soil or groundwater is measured and the receptor.

In the second case, a minimum lateral separation of 10 m is assumed between the location where the contaminated soil or groundwater is measured and the location of the surface water body. In cases where contamination is present within 10 m of a surface water body, a site-specific approach will be required (see AENV, 2009b).

Surface water quality guidelines protective of the above water uses are provided in Table 8. As noted in Section 10, insufficient data are available to calculate surface water guidelines for the Glycols protective of irrigation, wildlife or livestock watering, and accordingly, neither soil nor groundwater guidelines protective of these water uses could be calculated.

13.1 Soil Remediation Guidelines

Soil remediation guidelines for groundwater pathways were calculated using the model and equations from AENV (2009a)

13.1.1 Model Assumptions

Assumptions implicit in the model include the following:

- the soil is physically and chemically homogeneous;
- moisture content is uniform throughout the unsaturated zone;
- infiltration rate is uniform throughout the unsaturated zone;
- decay of the contaminant source is not considered (*i.e.*, infinite source mass);
- contaminant is not present as a free phase product;
- maximum possible concentration in the leachate is equivalent to the solubility limit of the chemical in water under the defined site conditions;

- the groundwater aquifer is unconfined;
- groundwater flow is uniform and steady;
- co-solubility and oxidation/reduction effects are not considered;
- attenuation of the contaminant in the saturated zone is assumed to be one dimensional with respect to sorption-desorption, dispersion, and biological degradation;
- dispersion in groundwater is assumed to occur in the longitudinal and transverse directions only and diffusion is not considered;
- mixing of the leachate with the groundwater is assumed to occur through mixing of leachate and groundwater mass fluxes; and
- dilution of the plume by groundwater recharge down-gradient of the source is not considered.

13.1.2 Guideline Calculation

The soil remediation guideline protective of groundwater uses is calculated in the same way for both groundwater uses noted at the start of this section, using the corresponding surface water quality guideline (Table 8) as the starting point for each. However, as noted above, the lateral offset between the point at which the contaminated soil is measured and the surface water body (parameter "x" in the equation for DF4 below) is assumed to be 10 m for aquatic life, and 0 m for human drinking water.

The model considers four processes:

1. partitioning from soil to leachate;
2. transport of leachate from base of contamination to water table;
3. mixing of leachate and groundwater; and,
4. groundwater transport down-gradient to a discharge point.

For each of these four processes, a dilution factor was calculated (DF1 through DF4, respectively). DF1 has units of (mg/kg)/(mg/L) or L/kg. The other three dilution factors are dimensionless [units of (mg/L)/(mg/L)]. The overall dilution factor is used to calculate the soil concentration that is protective of groundwater using the following equations:

$$SQG_{GR} = SWQG \times DF$$

$$DF = DF1 \times DF2 \times DF3 \times DF4$$

where:

SQG_{GR}= soil quality guideline protective of groundwater pathways (mg/kg)

SWQG= corresponding surface water quality guideline (drinking water or aquatic life) (mg/L)

DF = overall dilution factor (L/kg)
DF1 = dilution factor for process 1 (L/kg)
DF2 = dilution factor for process 2 (dimensionless)
DF3 = dilution factor for process 3 (dimensionless)
DF4 = dilution factor for process 4 (dimensionless)

Dilution Factor 1

Dilution factor 1 (DF1) is the ratio of the concentration of a contaminant in soil to the concentration in leachate that is in contact with the soil. This "dilution factor" represents the three phase partitioning between contaminant sorbed to soil, contaminant dissolved in pore water (*i.e.*, as leachate), and contaminant present as soil vapour. DF1 is calculated using the following equation:

$$DF1 = K_{oc} \times f_{oc} + \frac{(\theta_w + H' \times \theta_a)}{\rho_b}$$

where:

DF1 = dilution factor 1 (L/kg)
K_{oc} = organic carbon-water partition coefficient (L/kg)
f_{oc} = fraction organic carbon (g/g)
θ_w = water filled porosity (dimensionless)
H' = dimensionless Henry's Law constant (dimensionless)
θ_a = air filled porosity (dimensionless)
ρ_b = dry soil bulk density (g/cm^3)

Dilution Factor 2

Dilution factor 2 (DF2) is the ratio of the concentration of a contaminant in leachate that is in contact with the soil, to the concentration in pore water just above the groundwater table. DF2 takes the value 1.00 (*i.e.,* no dilution) for generic guidelines because it is assumed at Tier 1 that the contaminated soil extends down to the water table. Note that DF2 can be calculated on a site-specific basis at Tier 2 (AENV, 2009b).

Dilution Factor 3

Dilution factor 3 (DF3) is the ratio of the concentration of a chemical in pore water just above the groundwater table, to the concentration in groundwater beneath the source. This dilution factor reflects a decrease in concentration as leachate mixes with uncontaminated groundwater. DF3 is a function of groundwater velocity, infiltration rate, source length, and mixing zone thickness. The mixing zone thickness is calculated as being due to two processes: i) mixing due to dispersion, and ii) mixing due to infiltration rate. The equations used are as follows:

$$DF3 = 1 + \frac{Z_d \times V}{I \times X}$$

$$Z_d = r + s$$

$$r = 0.01 \times X$$

$$s = d_a \left\{ 1 - exp\left(\frac{-2.178 \times X \times I}{V \times d_a} \right) \right\}$$

$$V = K \times i$$

where:

DF3	=	dilution factor 3 (dimensionless)
Z_d	=	average thickness of mixing zone (m)
V	=	Darcy velocity in groundwater (m/year)
I	=	infiltration rate (m/year)
X	=	length of contaminated soil (m)
r	=	mixing depth due to dispersion (m)
s	=	mixing depth due to infiltration rate (m)
d_a	=	unconfined aquifer thickness (m)
K	=	aquifer hydraulic conductivity (m/year)
i	=	lateral hydraulic gradient in aquifer (dimensionless)

Note that the parameter Z_d takes the fixed value of 2 m for the drinking water pathway, but is calculated for all other pathways.

Dilution Factor 4

Dilution factor 4 (DF4) accounts for the processes of dispersion and biodegradation as groundwater travels downgradient from beneath the source of contamination, and is the ratio of the concentration of a chemical in groundwater beneath the source, to the concentration in groundwater at a distance of 10 m (at Tier 1 for aquatic life) downgradient of the source. Consistent with AENV (2009a), the time independent version of the equation to calculate DF4 was used:

$$DF4 = \frac{2}{\exp(A) \times [erf(C) - erf(D)]}$$

$$A = \frac{x}{2D_x}\left\{1-\left(1+\frac{4L_sD_x}{v}\right)^{1/2}\right\}$$

$$C = \frac{y+Y/2}{2\left(D_y x\right)^{1/2}}$$

$$D = \frac{y-Y/2}{2\left(D_y x\right)^{1/2}}$$

$$L_s = \frac{0.6931}{t_{1/2s}} \times \exp\left(-0.07d\right)$$

$$v = \frac{V}{\theta_t R_s}$$

$$R_s = 1 + \frac{\rho_b K_{oc} f_{oc}}{\theta_t}$$

$$D_x = 0.1x$$

$$D_y = 0.01x$$

where:

DF4 = dilution factor 4 (dimensionless)
erf = the error function
A = dimensionless group A (dimensionless)
C = dimensionless group C (dimensionless)
D = dimensionless group D (dimensionless)
x = distance to source (10 m, aquatic life and wildlife watering, 0 m other water uses)
D_x = dispersivity in the direction of groundwater flow (m)
L_s = decay constant (1/year)
v = velocity of the contaminant (m/year)
y = distance to receptor perpendicular to groundwater flow (m)
Y = source width (m)
D_y = dispersivity perpendicular to the direction of groundwater flow (m)
$t_{1/2s}$ = decay half-life of contaminant in saturated zone of aquifer (years)
d = water table depth (m)

V = Darcy velocity in groundwater (m/year)
θ_t = total soil porosity (dimensionless)
R_s = retardation factor in saturated zone (dimensionless)
ρ_b = dry soil bulk density (g/cm^3)
K_{oc} = organic carbon partition coefficient (mL/g)
f_{oc} = fraction organic carbon (g/g)

Aquatic Life
Substituting appropriate values from Tables 3, 5, 6, and 8 into this equation and rounding to 1 significant figure with a 5 or 0 in the second figure gives values of:

- 65 mg/kg (DEG, coarse soil; Table 9);
- 2,000 mg/kg (DEG, fine soil; Table 10);
- 200 mg/kg (TEG, coarse soil; Table 11); and,
- 10,000 mg/kg (TEG, fine soil; Table 12).

Protection of Domestic Use Aquifer
Substituting appropriate values from Tables 3, 5, 6, and 8 into this equation and rounding to 1 significant figure with a 5 or 0 in the second figure gives values of:

- 15 mg/kg (DEG, coarse soil; Table 9);
- 10 mg/kg (DEG, fine soil; Table 10);
- 150 mg/kg (TEG, coarse soil; Table 11); and,
- 100 mg/kg (TEG, fine soil; Table 12).

13.2 Groundwater Remediation Guidelines

Groundwater remediation guidelines for groundwater pathways were calculated using the model and equations from AENV (2009a).

13.2.1 Potable Groundwater

If contaminated groundwater is considered a domestic use aquifer, there is no offset assumed between contamination and a potential future water well, and therefore the Source Guidance Value for Groundwater (DEG = 6 mg/L; TEG = 60 mg/L) applies directly to groundwater (Tables 13 and 14).

13.2.2 Aquatic Life

Assumptions implicit in the model include the following:

- the soil/aquifer material in the saturated zone is physically and chemically homogeneous;
- decay of the contaminant source is not considered (*i.e.*, infinite source mass);
- the contaminant is not present as a free phase product;
- groundwater flow is uniform and steady;
- co-solubility and oxidation/reduction effects are not considered;
- dispersion is assumed to occur in the longitudinal and transverse directions only and diffusion is not considered; and,
- dilution of the plume by groundwater recharge down-gradient of the source is not considered.

Guideline Calculation

The groundwater remediation guideline protective of aquatic life is calculated using the following equations.

$$GWQG_{GR} = SWQG \times DF4$$

where:
$GWQG_{GR}$= groundwater quality guideline protective of aquatic life (mg/L)
$SWQG_{FL}$= surface water quality guideline protective of aquatic life (mg/L)
DF4 = dilution factor for process 4 (L/kg)

Dilution factor 4 is calculated in the same way as described in Section 13.1.2

Substituting appropriate values from Tables 3, 5, 6, and 8 into this equation and rounding to 1 significant figure with a 5 or 0 in the second figure gives values of:

- 200 mg/L (DEG, coarse soil; Table 13);
- 4,000 mg/L (DEG, fine soil; Table 13);
- 550 mg/L (TEG, coarse soil; Table 14); and,
- 25,000 mg/L (TEG, fine soil; Table 14).

14. REFERENCES

(See also References in Appendices A, B, and C)

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TABLES

Table 1. Common Synonyms and Trade Names for the Glycols

Diethylene Glycol	Triethylene Glycol	Tetraethylene Glycol
DEG	TEG	TREG
2,2'-oxybisethanol	1,2-bis(2-hydroxyethoxy)-ethane	3,6,9-trioxaundecan-1,11-diol
2,2'-dihydroxydiethyl ether	2,2'-(1,2-ethanediylbis-(oxy))bisethanol	ethanol, 2,2'-[oxybis(2,1-ethanediyloxy)]bis-
2,2'-dihydroxyethyl ether	2,2'-ethylenedioxybis(ethanol)	tetraethylene glycol
bis(2-Hydroxyethyl) ether	3,6-dioxa-1,8-octanediol	tetraglycol
2-hydroxyethyl ether	3,6-dioxaoctane-1,8-diol	
3-oxa-1,5-pentanediol	di-beta-hydroxyethoxyethane	
3-oxapentane-1,5-diol	ethylene glycol-bis-(2-hydroxyethyl)ether	
beta,beta'-dihydroxydiethyl ether	triethylene glycol	
diethylene glycol	trigen	
dihydroxyethylether	triglycol	
Dissolvant APV	trigol	
Brecolane NDG		
Carbitol		
Deactivator E		
Deactivator H		
ethylene diglycol		
glycol ether		
TL4N		

Table 2. Physical and Chemical Properties for the Glycols

Property	Units	DEG	TEG	TREG	Source
Formula	-----	$C_4H_{10}O_3$	$C_6H_{14}O_4$	$C_8H_{18}O_3$	1
CAS number	-----	111-46-6	112-27-6	112-60-7	1
Molecular weight	g/mole	106.1	150.2	164.2	2
Acid dissociation constant (pK_a)	-----	na	14.50	na	2
Melting point	°C	-10	-4	-6	2
Boiling point	°C	245	287	314	2
Specific gravity (at 20/4 °C)	g/cm^3	1.118	1.125	1.125	2
Vapour pressure (at 20 °C)	Pa	<1.3	<0.13	<1.3	2
Solubility (at 25 °C)	mg/L	miscible	miscible	miscible	1
Dimensionless Henry's law constant	-----	na[a]	5.3×10^{-9}	na	2
Organic carbon partition coefficient (K_{oc})	log	-1.74	-2.29	na	3,2
n-Octanol-water partition coefficient (K_{ow})	log	-1.98	-2.08	na	2

[a] - TEG Henry's law constant used to calculate DEG guidelines

Sources:

[1]CRC (1996)

[2]Verschueren (1983)

[3]DEG calculated from K_{ow} using Baker et al. (1997) equation provided in Boethling and Mackay (2000) (Table 8.1)

na = not available

Table 3. Chemical-Specific Parameter Values for DEG and TEG

Parameter	Unit	DEG	TEG	Rationale
Human Toxicity				
Tolerable Daily Intake (oral exposure)	mg/kg-bw/day	0.5	5	see Section 6.6
Tolerable Concentration (inhalation exposure)	mg/m^3	na	na	negligible vapour pressure
Human Background Exposure				
Estimated daily intake	mg/kg-bw/day	0	0	see Section 2.6
Ambient air concentration	mg/m^3	0	0	see Section 2.6
Background soil concentration	mg/kg	0	0	see Section 2.6
Soil allocation factor	-	0.25	0.25	see Section 9.1
Water allocation factor	-	0.25	0.25	see Section 9.1
Human Adsorption				
Adsorption factor - gut	-	1.0	1.0	assumed
Adsorption factor - gut	-	1.0	1.0	assumed
Adsorption factor - gut	-	1.0	1.0	assumed
Chemical and Physical Properties				
Soil Organic Carbon/Water Partition Coefficient (Koc)	L/kg	0.018	0.0051	see Table 2
Dimensionless Henry's law coeffcient	(mg/L)/(mg/L)	5.3×10^{-9}	5.3×10^{-9}	assumed; see Table 2
Dynamic viscosity of vapour	g/cm.s	1.73×10^{-4}	1.73×10^{-4}	AENV (2009a)
Diffusion coefficient in air	cm^2/s	na	na	negligible vapour pressure
Degradation				
Degradation half life (saturated)	days	250	175	see Section 3.5.3

Notes:
na = not applicable

Table 4. Human Receptor Characteristics

Parameter	Symbol	Unit	Toddler	Adult
Body Weight	BW	kg	16.5	70.7
Air Inhalation Rate	IR	m^3/d	9.3	15.8
Soil (Dust) Inhalation Rate	IR_S	kg/d	7.1×10^{-9}	1.2×10^{-8}
Water Ingestion Rate	WIR	L/d	0.6	1.5
Soil Ingestion Rate	SIR	kg/d	0.00008	0.00002
Skin Surface Area				
- Hands	SA_H	m^2	0.043	0.089
- Other	SA_O	m^2	0.258	0.25
Dermal Loading to Skin				
- Hands	DL_H	kg/m^2-event	0.001	0.001
- Other	DL_O	kg/m^2-event	0.0001	0.0001
Dermal Exposure Frequency	EF	events/d	1	1
Exposure Term, agricultural and residential/parkland	ET	-	1	1
Exposure Term, commercial and industrial	ET	-	0.2747	0.2747
Exposure Term, agricultural and residential/parkland	ET_1	-	1	1
Exposure Term, commercial and industrial	ET_1	-	0.6593	0.6593
Exposure Term, agricultural and residential/parkland	ET_2	-	1	1
Exposure Term, commercial and industrial	ET_2	-	0.4167	0.4167

Notes:

All parameter values from AENV (2009a)

Table 5. Soil and Hydrogeological Parameters

Parameter	Symbol	Unit	Fine Soil	Coarse Soil
Soil Bulk Density	ρ_B	kg/L	1.4	1.7
Soil Total Porosity	θ_t	cm^3/cm^3	0.47	0.36
Soil Moisture-Filled Porosity	θ_w	cm^3/cm^3	0.168	0.119
Soil Vapour-Filled Porosity	θ_a	cm^3/cm^3	0.302	0.241
Soil Vapour-Filled Porosity in Floor Cracks	θ_a	cm^3/cm^3	0.47	0.36
Gravimetric Water Content	MC	g/g	0.12	0.07
Fraction of Organic Carbon	f_{oc}	mass/mass	0.005	0.005
Saturated Hydraulic Conductivity	K	m/y	32	320
Hydraulic Gradient	i	m/m	0.028	0.028
Recharge (Infiltration) Rate	I	m/y	0.012	0.06
Soil Permeability to Vapour Flow	k_v	cm^2	10^{-9}	$6x10^{-8}$

Notes:

All parameter values from AENV (2009a)

Table 6. Site Characteristics

Parameter	Symbol	Unit	Value
Contaminant Source Width	Y	m	10
Contaminant Source Length	X	m	10
Contaminant Source Depth	Z	m	3
Distance to Surface Water	x	m	10
Distance to Potable Water User	x	m	0
Distance to Agricultural Water User	x	m	0
Distance from Contamination to Building Slab	L_T	cm	30
Depth to Groundwater (water table)	d	m	3
Depth of unconfined aquifer	d_a	m	5

Notes:
All parameter values from AENV (2009a)

Table 7. Building Parameters

Parameter	Symbol	Unit	Residential Basement	Residential Slab-on-Grade	Commercial Slab-on-Grade
Building Length	L_B	cm	1,225	1,225	2,000
Building Width	W_B	cm	1,225	1,225	1,500
Building Height (including basement)	H_B	cm	360	360	300
Area of Substructure	A_B	cm^2	$2.7x10^6$	$1.5x10^6$	$3.0x10^6$
Thickness of Floor Slab	L_{crack}	cm	11.25	11.25	11.25
Depth of Floor Slab Below Ground	Z_{crack}	cm	244	11.25	11.25
Distance from Source to Slab:	L_T	cm			
surface soil			30	30	30
subsoil			30	139	139
Crack Area	A_{crack}	cm^2	994.5	994.5	1,846
Crack Length	X_{crack}	cm	4,900	4,900	7,000
Air Exchange Rate	ACH	exch/hr	0.5	0.5	0.9
Pressure Differential	ΔP	$g/cm.s^2$	40	40	20

Notes:

All parameter values from AENV (2009a)

Table 8. Surface Water Quality Guidelines for DEG and TEG

Water Use	DEG (mg/L)	TEG (mg/L)
Human drinking water ("Source Guidance Value for Groundwater")	6	60
Freshwater aquatic life	150	350
Irrigation [1]	n/c	n/c
Livestock watering [2]	n/c	n/c
Wildlife watering [3]	n/c	n/c

Notes:

n/c = not calculated

1. *guideline protective of irrigation not calculated due to lack of toxicity data relevant to irrigation.*
2. *guideline not calculated due to the lack of toxicity information for livestock species.*
3. *guideline not calculated due to the lack of toxicity information for wildlife species.*

Table 9. Soil Remediation Guidelines for DEG - Coarse Soil

	Guideline Value (mg/kg)				
Land Use:	**Natural Area**	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Overall Guideline	**15**	**15**	**15**	**15**	**15**
Human Exposure Pathways					
Direct soil contact	n/a	15,000	15,000	20,000	100,000
Vapour inhalation	n/a	n/c	n/c	n/c	n/c
Protection of domestic use aquifer	15	15	15	15	15
Produce, milk and meat check [1]	n/c	n/c	n/c	n/c	n/c
Off-site migration [2]	n/a	n/a	n/a	200,000	200,000
Ecological Exposure Pathways					
Direct soil contact	1,000	1,000	1,000	1,500	1,500
Nutrient and Energy cycling check [3]	n/c	n/c	n/c	n/c	n/c
Livestock soil and food ingestion [4]	n/c	n/c	n/c	n/c	n/c
Protection of freshwater aquatic life	65	65	65	65	65
Off-site migration [2]	n/a	n/a	n/a	15,000	15,000

Notes:

n/a = exposure pathway not applicable in this scenario.

n/c = not calculated

1. *produce, meat and milk check not calculated - glycols not expected to accumulate in produce, milk, or meat.*
2. *offsite migration not considered a concern given the degradability of glycols in conditions likely to be found at surface (Section 3.5.3).*
3. *Nutrient and energy cycling check not calculated - insufficient data*
4. *Livestock soil and food ingestion not expected to be a concern, glycols expected to be lost rapidly from surface soil, and not accumulate into fodder.*

Table 10. Soil Remediation Guidelines for DEG - Fine Soil

	Guideline Value (mg/kg)				
Land Use:	**Natural Area**	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Overall Guideline	**10**	**10**	**10**	**10**	**10**
Human Exposure Pathways					
Direct soil contact	n/a	15,000	15,000	20,000	100,000
Vapour inhalation	n/a	n/c	n/c	n/c	n/c
Protection of domestic use aquifer	10	10	10	10	10
Produce, milk and meat check [1]	n/c	n/c	n/c	n/c	n/c
Off-site migration [2]	n/a	n/a	n/a	200,000	200,000
Ecological Exposure Pathways					
Direct soil contact	1,000	1,000	1,000	1,500	1,500
Nutrient and Energy cycling check [3]	n/c	n/c	n/c	n/c	n/c
Livestock soil and food ingestion [4]	n/c	n/c	n/c	n/c	n/c
Protection of freshwater aquatic life	2,000	2,000	2,000	2,000	2,000
Off-site migration [2]	n/a	n/a	n/a	15,000	15,000

Notes:

n/a = exposure pathway not applicable in this scenario.

n/c = not calculated

1. *produce, meat and milk check not calculated - glycols not expected to accumulate in produce, milk, or meat.*
2. *offsite migration not considered a concern given the degradability of glycols in conditions likely to be found at surface (Section 3.5.3).*
3. *Nutrient and energy cycling check not calculated - insufficient data*
4. *Livestock soil and food ingestion not expected to be a concern, glycols expected to be lost rapidly from surface soil, and not accumulate into fodder.*

Table 11. Soil Remediation Guidelines for TEG - Coarse Soil

	Guideline Value (mg/kg)				
Land Use:	**Natural Area**	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Overall Guideline	**150**	**150**	**150**	**150**	**150**
Human Exposure Pathways					
Direct soil contact	n/a	150,000	150,000	200,000	ngr
Vapour inhalation	n/a	n/c	n/c	n/c	n/c
Protection of domestic use aquifer	150	150	150	150	150
Produce, milk and meat check [1]	n/c	n/c	n/c	n/c	n/c
Off-site migration [2]	n/a	n/a	n/a	ngr	ngr
Ecological Exposure Pathways					
Direct soil contact	5,000	5,000	5,000	7,000	7,000
Nutrient and Energy cycling check [3]	n/c	n/c	n/c	n/c	n/c
Livestock soil and food ingestion [4]	n/c	n/c	n/c	n/c	n/c
Protection of freshwater aquatic life	200	200	200	200	200
Off-site migration [2]	n/a	n/a	n/a	70,000	70,000

Notes:

n/a = exposure pathway not applicable in this scenario.

n/c = not calculated

ngr = no guideline required - calculated value $>10^6$ mg/kg

1. *produce, meat and milk check not calculated - glycols not expected to accumulate in produce, milk, or meat.*
2. *offsite migration not considered a concern given the degradability of glycols in conditions likely to be found at surface (Section 3.5.3).*
3. *Nutrient and energy cycling check not calculated - insufficient data*
4. *Livestock soil and food ingestion not expected to be a concern, glycols expected to be lost rapidly from surface soil, and not accumulate into fodder.*

Table 12. Soil Remediation Guidelines for TEG - Fine Soil

	Guideline Value (mg/kg)				
Land Use:	**Natural Area**	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Overall Guideline	**100**	**100**	**100**	**100**	**100**
Human Exposure Pathways					
Direct soil contact	n/a	150,000	150,000	200,000	ngr
Vapour inhalation	n/a	n/c	n/c	n/c	n/c
Protection of domestic use aquifer	100	100	100	100	100
Produce, milk and meat check [1]	n/c	n/c	n/c	n/c	n/c
Off-site migration [2]	n/a	n/a	n/a	ngr	ngr
Ecological Exposure Pathways					
Direct soil contact	5,000	5,000	5,000	7,000	7,000
Nutrient and Energy cycling check [3]	n/c	n/c	n/c	n/c	n/c
Livestock soil and food ingestion [4]	n/c	n/c	n/c	n/c	n/c
Protection of freshwater aquatic life	10,000	10,000	10,000	10,000	10,000
Off-site migration [2]	n/a	n/a	n/a	70,000	70,000

Notes:

n/a = exposure pathway not applicable in this scenario.

n/c = not calculated

1. *produce, meat and milk check not calculated - glycols not expected to accumulate in produce, milk, or meat.*
2. *offsite migration not considered a concern given the degradability of glycols in conditions likely to be found at surface (Section 3.5.3).*
3. *Nutrient and energy cycling check not calculated - insufficient data*
4. *Livestock soil and food ingestion not expected to be a concern, glycols expected to be lost rapidly from surface soil, and not accumulate into fodder.*

Table 13. Groundwater Remediation Guidelines for DEG

	Guideline Value (mg/L)				
Land Use:	**Natural Area**	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Lowest Guideline (Coarse)	**6**	**6**	**6**	**6**	**6**
Lowest Guideline (Fine)	**6**	**6**	**6**	**6**	**6**
Water Use					
Potable groundwater	6	6	6	6	6
Vapour inhalation from groundwater[1]					
Coarse soil	n/a	n/c	n/c	n/c	n/c
Fine soil	n/a	n/c	n/c	n/c	n/c
Groundwater protective of eco-contact[2]					
Coarse soil	n/c	n/c	n/c	n/c	n/c
Fine soil	n/c	n/c	n/c	n/c	n/c
Groundwater protective of freshwater aquatic life					
Coarse soil	200	200	200	200	200
Fine soil	4,000	4,000	4,000	4,000	4,000
Groundwater used for irrigation[3]	n/c	n/c	n/c	n/c	n/c
Groundwater used for livestock watering[4]	n/c	n/c	n/c	n/c	n/c
Groundwater used for wildlife watering[5]	n/c	n/c	n/c	n/c	n/c

Notes:

n/a = water use not applicable in this scenario.

n/c = not calculated

1. *pathway not a concern - glycols have negligible vapour pressure*
2. *see section 12.1.2*
3. *groundwater protective of irrigation not calculated due to lack of toxicity data relevant to irrigation.*
4. *Livestock watering groundwater guideline not calculated due to the lack of toxicity information for livestock species.*
5. *Wildlife watering groundwater guideline not calculated due to the lack of toxicity information for wildlife species.*

Table 14. Groundwater Remediation Guidelines for TEG

	Guideline Value (mg/L)				
Land Use:	**Natural Area**	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Lowest Guideline (Coarse)	**60**	**60**	**60**	**60**	**60**
Lowest Guideline (Fine)	**60**	**60**	**60**	**60**	**60**
Water Use					
Potable groundwater	60	60	60	60	60
Vapour inhalation from groundwater[1]					
Coarse soil	n/a	n/c	n/c	n/c	n/c
Fine soil	n/a	n/c	n/c	n/c	n/c
Groundwater protective of eco-contact[2]					
Coarse soil	n/c	n/c	n/c	n/c	n/c
Fine soil	n/c	n/c	n/c	n/c	n/c
Groundwater protective of freshwater aquatic life					
Coarse soil	550	550	550	550	550
Fine soil	25,000	25,000	25,000	25,000	25,000
Groundwater used for irrigation[3]	n/c	n/c	n/c	n/c	n/c
Groundwater used for livestock watering[4]	n/c	n/c	n/c	n/c	n/c
Groundwater used for wildlife watering[5]	n/c	n/c	n/c	n/c	n/c

Notes:

n/a = water use not applicable in this scenario.

n/c = not calculated

1. *pathway not a concern - glycols have negligible vapour pressure*
2. *see section 12.1.2*
3. *groundwater protective of irrigation not calculated due to lack of toxicity data relevant to irrigation.*
4. *Livestock watering groundwater guideline not calculated due to the lack of toxicity information for livestock species.*
5. *Wildlife watering groundwater guideline not calculated due to the lack of toxicity information for wildlife species.*

FIGURES

Figure 1. Major Uses of DEG and TEG

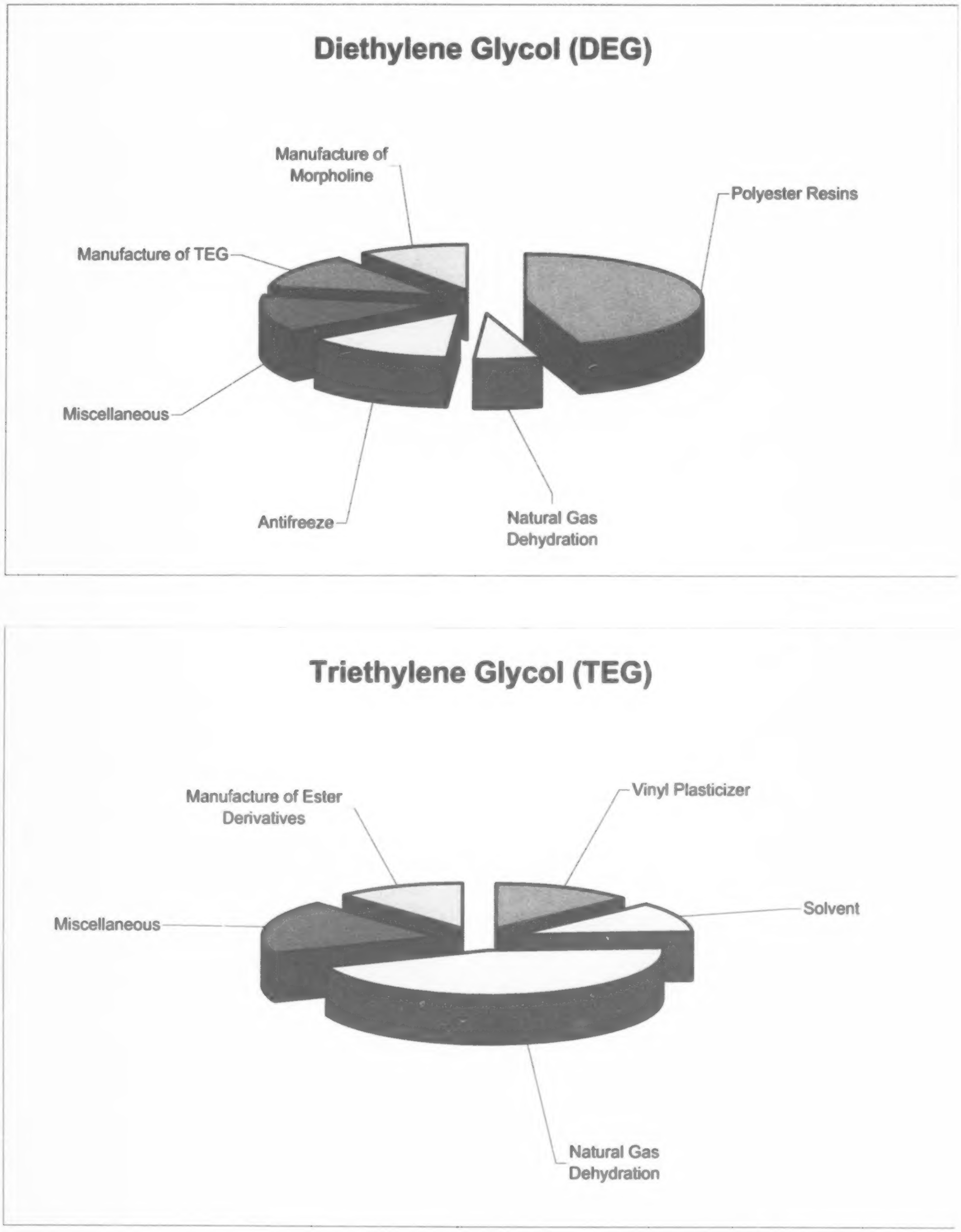
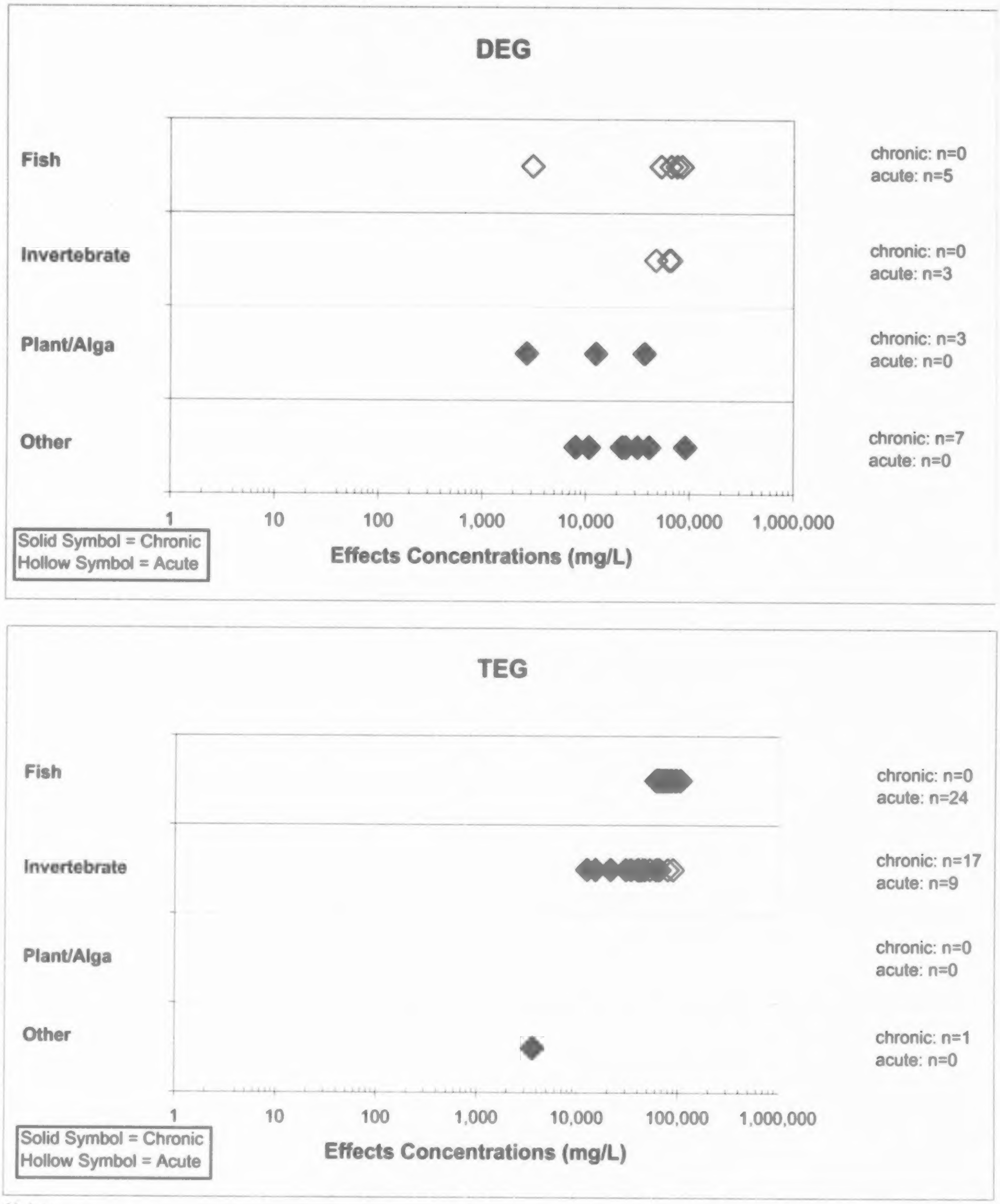


Figure 2. Effects Concentrations of DEG and TEG to Freshwater Aquatic Organisms



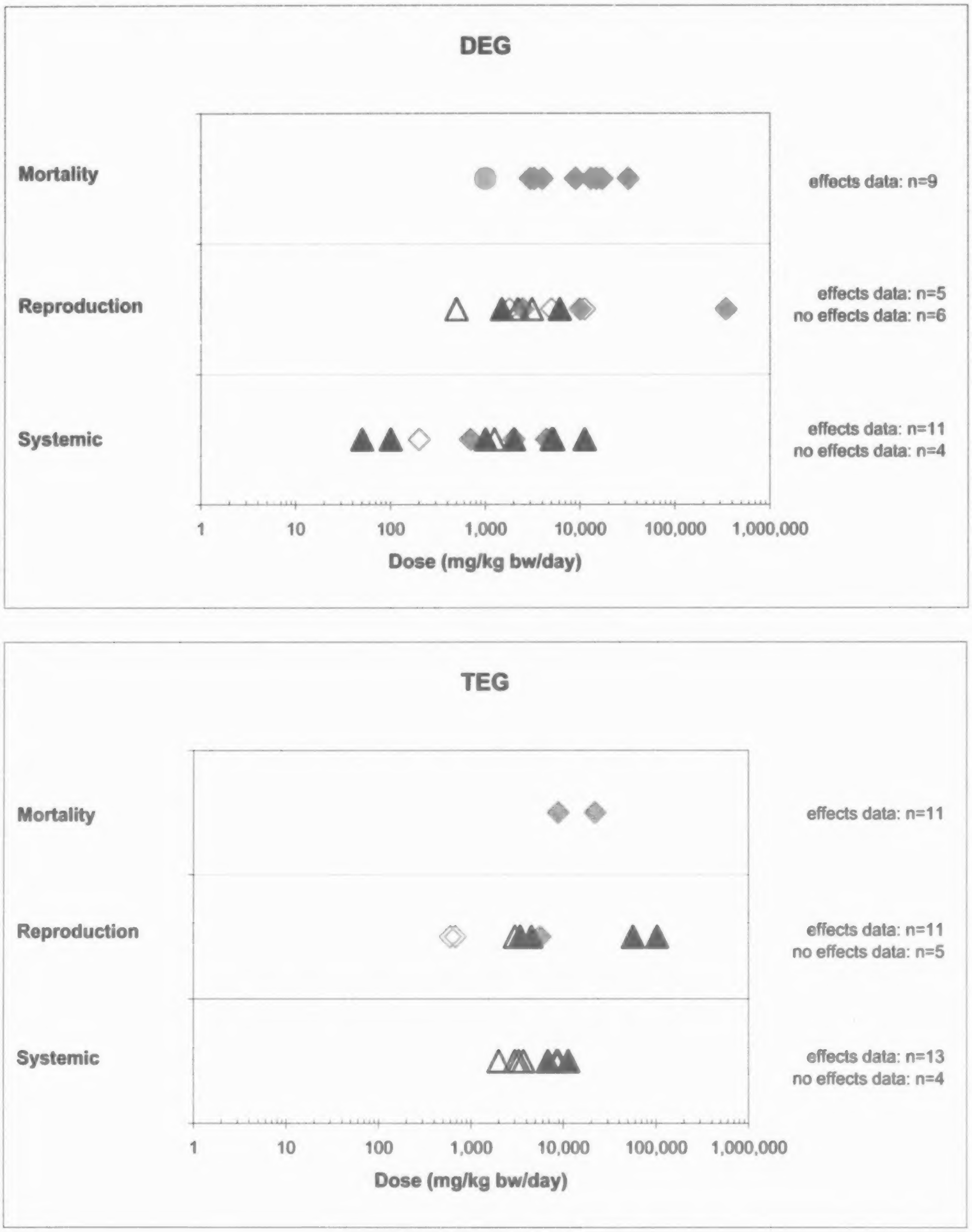
Notes:

Solid Symbol = Chronic

Hollow Symbol = Acute

Only data of Primary or Secondary quality included.

Figure 3. Oral Toxicity of DEG and TEG to Mammalian Species



Notes:
Solid Symbol = Effects
Hollow Symbol = No Effect

Diamond = Animal Study, Acute
Triangle = Animal Study, (Sub-)Chronic
Circle = Human Data

APPENDIX A

DEG
DEGRADATION AND TOXICITY DATA

Table A-1 Summary of Available Data on DEG Biodegradation
Table A-2 Toxicity of DEG to Freshwater Aquatic Life
Table A-3 Toxicity of DEG to Marine Aquatic Life
Table A-4 Toxicity of DEG to Terrestrial Plants
Table A-5 Toxicity of DEG to Terrestrial Invertebrates
Table A-6 Toxicity of DEG to Mammalian Species

Table A-1. Summary of Available Data on DEG Biodegradation

Test Method	Test Duration	Aerobic/ Anaerobic	Initial Compound Concentration	% Removed	Inoculum or Medium	Interpreted Half-Life	Rates / Comments	Reference
Studies Conducted under Unamended Conditions								
batch cultures; GC/MS analysis	32 days	aerobic	100 ppm	75%	distilled water	20 days	Similar rates for aerobic, anaerobic, and abiotic. Degradation interpreted to be abiotic in all cases.	Kaplan et al. (1982)
respirometry	90 days	aerobic	200 mg/kg	78%	Alberta Soil	16 days	lag period: 5.6 days, hyperbolic kinetics; 25C	Sorensen et al. (2000)
respirometry	90 days	aerobic	1,000 mg/kg	54%	Alberta Soil	143 days	lag period: 3.4 days, hyperbolic kinetics; 25C	Sorensen et al. (2000)
respirometry	90 days	aerobic	200 mg/kg	88%	New Mexico Soil	33 days	lag period: 6.1 days; 25C	Sorensen et al. (2000)
respirometry	90 days	aerobic	1,000 mg/kg	29%	New Mexico Soil	208 days	lag period: 4.7 days; 25C	Sorensen et al. (2000)
respirometry	90 days	aerobic	200 mg/kg	97%	Louisiana Soil	50 days	lag period: 19.6 days; 25C; slow kinetics may indicate toxicity	Sorensen et al. (2000)
respirometry	90 days	aerobic	1,000 mg/kg	33%	Louisiana Soil	250 days	lag period: 39.9 days; 25C; slow kinetics may indicate toxicity	Sorensen et al. (2000)
Other Studies								
degradability test	5 days	aerobic	nv	nv	nv	nd	"extensive" degradation ofter 5 days	Haines and Alexander (1975)
batch cultures; GC/MS analysis	32 days	both	100 ppm	75%	acclimated sludge and nutrient broth	20 days	Similar rates for aerobic, anaerobic, and abiotic. Degradation interpreted to be abiotic in all cases.	Kaplan et al. (1982)
degradability test	3-14 days	aerobic	2-10 mg/L	nv	river water	nd	biodegdatation in some waters was complete after 4 days at both 4 and 20C, in other waters no measurable degradation occurred in 14 days at either temperature.	Evans and David (1974)
degradability test	8 hours	aerobic	333 mg/L	0%	acclimated sludge	nd	no reduction in BOD or COD noted	Hatfield (1957)
degradability test	nv	aerobic	nv	nv	TEG-adapted gram negative bacterium	nd	bacterium could grow on DEG but DEG not utilized if alternative carbon source available	Fincher and Payne (1962)
degradability test	nv	aerobic	nv	nv	adapted cells	nd	DEG consumed 114% of theoretical oxygen consumption, indicating degradation of DEG; oxidation rate decreased TEG>DEG>EG	Fincher and Payne (1962)
degradability test	nv	aerobic	nv	nv	Glucobacter oxydans	nd	positive result for glycol oxidation	Kersters and DeLey (1963)
degradability test	nv	aerobic	nv	nv	nv	nd	Degradation by the bacterium genus *Acinetobacter* noted	Jones and Watson (1976)

Table A-1. Summary of Available Data on DEG Biodegradation

Test Method	Test Duration	Aerobic/ Anaerobic	Initial Compound Concentration	% Removed	Inoculum or Medium	Interpreted Half-Life	Rates / Comments	Reference
degradability test	nv	aerobic	200 mg/L (as COD)	95%	sludge	nd	rapid degradation	Pitter (1976)
degradability test	nv	aerobic	nv	nv	nv	nd	Degradation by the bacterium genus *Alcaligenes* noted	Harada and Nagashima (1975)
degradability test	nv	aerobic	nv	nv	nv	nd	Degraded by bacteria of genus *Acinetobacter* and *Pseudomonas,* but not *Flavobacterium.*	Jones and Watson (1976)
degradability test	nv	both	nv	nv	aerobic and anaerobic sludge	nd	Degradation observed, but appeared to be abiotic. Analysis by GC.	Kaplan et al. (1982)
degradability - methanogenic	nv	anaerobic	nv	nv	methanogenic conditions	nd	degradation observed under methanogenic conditions.	Dwyer and Tiedje (1983)
degradability - anaerobic	nv	aerobic	nv	nv	nv	nd	glycol metabolism by *Desulfovibrio desulphuricans.*	Dwyer and Tiedje (1983)
degradability test	24 hours	aerobic	2,100 mg/L	24%	bioreactor	nd	based on COD reduction.	ARCO Chemical Company (1990)
BOD reduction	5 days	aerobic	nv	1.3-10%	nv	nd	based on theoretical oxygen demand (ThOD)	Verschueren (2001)
BOD reduction	10 days	aerobic	nv	0-5.6%	nv	nd	based on theoretical oxygen demand (ThOD)	Verschueren (2001)
BOD reduction	15 days	aerobic	nv	9%	nv	nd	based on theoretical oxygen demand (ThOD)	Verschueren (2001)
BOD reduction	20 days	aerobic	nv	19-21%	nv	nd	based on theoretical oxygen demand (ThOD)	Verschueren (2001)
degradability	up to 60 days	aerobic	various	up to 97%	various	nd	49 data point s from 21 studies, most indicating significant biodegradation.	Verschueren (2001)

[a] Biochemical oxygen demand (BOD) is defined as parts of oxygen consumed per part of compound during degradation. This value is expressed as a percentage of the theoretical (ThOD) oxygen demand.

nv = not reported in the abstract and not verified in the literature search

nd = not determined

Table A-2. Toxicity of DEG to Freshwater Aquatic Life

Biota Type	Scientific Name	Common Name	Study Type	Test Duration	Concentration	Endpoint	Effect	Exposure Type	pH	Temperature	Chemical Analysis	Control Type	Reference
					mg/L					°C			
Primary Data													
vertebrate	*Oncorhynchus mykiss*	Rainbow trout	acute	96 h	52,800	LC50	Mortality	Static	7.4	22	measured	satisfactory	Ward et al. (1992)
vertebrate	*Oncorhynchus mykiss*	Rainbow trout	acute	96 h	66,000	LC50	Mortality	Static	7.7	15	measured	satisfactory	Vizon (2006)
vertebrate	*Pimephales promelas*	Fathead minnow	acute	96 h	75,200	LC50	Mortality	Flow Through	7.7	25	measured	satisfactory	Geiger et al. (1990)
vertebrate	*Pimephales promelas*	Fathead minnow	acute	96 h	84,100	LC50	Mortality	Static	8.1	22	measured	satisfactory	Ward et al. (1992)
invertebrate	*Daphnia magna*	Water flea	acute	48 h	47,200	LC50	Mortality	Static	7.7	22	measured	satisfactory	Ward et al. (1992)
invertebrate	*Daphnia magna*	Water flea	acute	48 h	63,000	LC50	Mortality	Static	8.0	20	measured	satisfactory	Vizon (2006)
invertebrate	*Hyalella azteca*	Amphipod	acute	96 h	66,000	LC50	Mortality	Static	7.9	23	measured	satisfactory	Vizon (2006)
plant/alga	*Selenastrum capricornutum*	green alga	chronic	14 d	37,000	IC50	Growth	Static	7.6	22	measured	satisfactory	Ward et al. (1992)
plant/alga	*Selenastrum capricornutum*	green alga	chronic	14 d	12,500	LOEC	Growth	Static	7.6	22	measured	satisfactory	Ward et al. (1992)
Secondary Data													
vertebrate	*Xenopus laevis*	Clawed toad	acute	48 h	3,065	LC50	Mortality	Static	nr	20	nominal	satisfactory	de Zwart and Slooff (1987)
plant/alga	*Scenedesmus quadricauda*	Green alga	chronic	7 d	2,700	IC03	Growth	Static	nr	27	nominal	satisfactory	Bringmann and Kuhn (1980a)
other	*Entosiphon sulcatum*	Flagellate euglenoid	chronic	72 h	10,745	IC03	Growth	Static	nr	26	nominal	satisfactory	Bringmann and Kuhn (1980a)
other	*Pseudomonas putida*	Bacterium	chronic	16 h	8,000	IC03	Growth	Static	nr	25	nominal	satisfactory	Bringmann and Kuhn (1980a)
other	*Tetrahymena pyriformis*	Ciliate protozoan	chronic	36 h	22,500	IC50	Growth	Static	nr	28	nominal	satisfactory	Sauvant et al. (1995b)
other	*Tetrahymena pyriformis*	Ciliate protozoan	chronic	9 h	24,400	IC50	Growth	Static	nr	28	nominal	satisfactory	Sauvant et al. (1995b)
other	*Tetrahymena pyriformis*	Ciliate protozoan	chronic	3 h	91,150	IC50	Growth	Static	nr	28	nominal	satisfactory	Sauvant et al. (1995a)
other	*Tetrahymena pyriformis*	Ciliate protozoan	chronic	6 h	41,000	IC50	Growth	Static	nr	28	nominal	satisfactory	Sauvant et al. (1995a)
other	*Tetrahymena pyriformis*	Ciliate protozoan	chronic	9 h	31,500	IC50	Growth	Static	nr	28	nominal	satisfactory	Sauvant et al. (1995a)
Unacceptable Data (Based on Unverifiable Control Information)													
other	*Anacystis aeruginosa*	Blue-green algae	chronic	8 d	1,700	nv	Growth	Static	nv	28	nominal	nv	Bringmann and Kuhn (1978b)
other	*Microcystis aeruginosa*	Blue-green algae	chronic	8 d	1,700	LOEC	Growth	Static	nv	nv	nominal	nv	Bringmann and Kuhn (1978a)

Table A-2. Toxicity of DEG to Freshwater Aquatic Life

Biota Type	Scientific Name	Common Name	Study Type	Test Duration	Concentration	Endpoint	Effect	Exposure Type	pH	Temperature	Chemical Analysis	Control Type	Reference
					mg/L					°C			
Data Excluded Due to Lack of Effects at Maximum Concention Tested													
vertebrate	*Gambusia affinis*	Western mosquitofish	acute	96 h	32,000	NOEC	Mortality	Static	8.2	23	nominal	nv	Wallen et al. (1957)
vertebrate	*Carassius auratus*	Goldfish	acute	24 h	>5,000	LC50	Mortality	Static	7	20	measured	satisfactory	Bridie et al (1979)
vertebrate	*Gambusia affinis*	Western mosquitofish	acute	24 h	>32,000	LC50	Mortality	Static	8.2	23	nominal	nv	Wallen et al. (1957)
vertebrate	*Gambusia affinis*	Western mosquitofish	acute	48 h	>32,000	LC50	Mortality	Static	8.2	23	nominal	nv	Wallen et al. (1957)
vertebrate	*Gambusia affinis*	Western mosquitofish	acute	96 h	>32,000	LC50	Mortality	Static	8.2	23	nominal	nv	Wallen et al. (1957)
vertebrate	*Lepomis macrochirus*	Bluegill	acute	96 h	>1,000	LC0	Mortality	Static	nv	20	nominal	satisfactory	Buzzell et al. (1968)
vertebrate	*Leuciscus idus melanotus*	Carp	acute	48 h	>10,000	LC50	Mortality	nv	nv	nv	nv	nv	Juhnke and Luedemann (1978)
invertebrate	*Daphnia magna*	Water flea	acute	24 h	>10,000	LC50	Mortality	Static	7.6	21	nominal	nv	Bringmann and Kuhn (1977)
invertebrate	*Daphnia magna*	Water flea	acute	24 h	>10,000	EC50	Behaviour	nv	nv	nv	nv	nv	Bringmann and Kuhn (1982)
plant/alga	*Chlorococcales*	Green algae order	acute	24 h	>1,000	EC10	similation Efficiei	Static	nv	nv	nv	nv	Krebs (1991)
other	*Chilomonas paramecium*	Cryptomonad	chronic	48 h	>4,000	nv	Growth	nv	6.9	20	nominal	nv	Bringmann et al. (1980b)
other	*Chilomonas paramecium*	Cryptomonad	NV	NV	>4,000	nv	Growth	nv	nv	nv	nv	nv	Bringmann and Kuhn (1981)
other	*Uronema parduczi*	Ciliate protozoan	NV	NV	>8,000	nv	Growth	nv	nv	nv	nv	nv	Bringmann and Kuhn (1981)
other	*Uronema parduczi*	Ciliate protozoan	acute	20 h	>8,000	nv	Growth	nv	6.9	nv	nv	nv	Bringmann and Kuhn (1980b)

Notes:

nv = not reported in the abstract and not verified in this literature search

nr = not reported in the paper.

Table A-3. Toxicity of DEG to Marine Aquatic Life

Biota Type	Scientific Name	Common Name	Study Type	Test Duration	Concentration	Endpoint	Effect	Exposure Type	pH	Temperature	Salinity	Chemical Analysis	Control Type	Reference
					mg/L					°C	ppt			
Primary Data														
vertebrate	*Pimephales promelas*	sheepshgead minnow	acute	96 h	62,100	LC50	Mortality	Static	8.1	22	11-16	m	s	Ward et al. (1992)
invertebrate	*Mysidopsis bahia*	mysid	acute	96 h	36,900	LC50	Mortality	Static	7.7	22	11-13	m	s	Ward et al. (1992)
plant/alga	*Skeletonema costatum*	green alga	chronic	14 d	22,600	IC50	Mortality	Static	7.6	22	nr	m	s	Ward et al. (1992)
Unacceptable or Unverified Data														
invertebrate	*Artemia salina*	Brine shrimp	acute	24 h	>10,000	LC50	Mortality	Static	nv	24	nv	n	nv	Price et al. (1974)

Notes:

nv = not reported in the abstract and not verified in this literature search

nr = not reported in the paper.

chemical analysis: m = measured, n = nominal

control type: c = concurrent; s = satisfactory

Table A-4. Toxicity of DEG to Terrestrial Plants

Scientific Name	Common Name	Effect Measurement	Concentration	Endpoint/ Response	Response Site	Test Duration	Media Type	Application Method	Chemical Analysis	Reference
						days				
Medicago sativa	Alfalfa	Length	1,297	IC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Medicago sativa	Alfalfa	Length	1,489	IC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Medicago sativa	Alfalfa	Dry Mass	2,533	IC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Medicago sativa	Alfalfa	Dry Mass	2,472	IC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Length	2,706	IC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Length	2,742	IC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Dry Mass	419	IC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Dry Mass	968	IC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Length	1,740	IC25	shoot	21	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Length	1,889	IC25	root	21	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Dry Mass	818	IC25	shoot	21	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Dry Mass	1,119	IC25	root	21	artificial soil	spiked	Y	Stantec (2006)

Notes:

values presented here are nominal - not corrected for analytical recovery.

Table A-5. Toxicity of DEG to Terrestrial Invertebrates

Scientific Name	Common Name	Effect Measurement	Concentration	Endpoint/Response	Test Duration	Media Type	Application Method	Chemical Analysis	Reference
					days				
Eisenia andrei	Earthworm	adult survival	10,974	LC50	35	artificial soil	spiked	Y	Stantec (2006)
Eisenia andrei	Earthworm	# progeny	7,697	IC25	63	artificial soil	spiked	Y	Stantec (2006)
Eisenia andrei	Earthworm	progeny wet mass	5,050	IC25	63	artificial soil	spiked	Y	Stantec (2006)
Eisenia andrei	Earthworm	progeny dry mass	4,842	IC25	63	artificial soil	spiked	Y	Stantec (2006)
Folsomia candida	Springtail	adult survival	15,689	LC50	28	artificial soil	spiked	Y	Stantec (2006)
Folsomia candida	Springtail	# progeny	5,341	IC25	28	artificial soil	spiked	Y	Stantec (2006)

Notes:

values presented here are nominal - not corrected for analytical recovery.

IC25/LC25 values presented where available, otherwise IC50/LC50 presented

Table A-6. Toxicity of DEG to Mammalian Species

Study Type	Species	Route	NOAEL/ NOAEC	LOAEL/ LOAEC/ LPTD	LD50/ LC50	Duration/ Exposure	Endpoint	Reference
Acute								
acute	cat	oral			3,300 mg/kg	single	N/R	Laug et al. (1939)
acute	dog	oral			9,000 mg/kg	single	N/R	Hanzlik et al. (1939)
acute	hamster	oral			>7,500 mg/kg	single	death	Yoshida et al. (1986)
acute	human	oral			1 gm/kg (LPLD)	single	N/R	Laug et al. (1939)
acute	human	oral				unknown	DEG in pharmaceuticals (21 patients died)	Pandya (1988)
acute	human	oral				unknown	kidney damage following ingestion of DEG in wine	van Leusen and Uges (1987)
acute	mouse	oral			>20,000	single	death	Laug et al. (1939); Meyer and Sturmer (1952)
acute	rabbit	oral			4,400 mg/kg	single	respiration effects, hypothermia, coma	Laug et al. (1939)
acute	rabbit, guinea pig, dog	oral			4,000-17,000 mg/kg	single	death	Laug et al. (1939);Smyth et al. (1941).
acute	rat	oral		0.7 g/kg		single	increased LDH in urine	Freundt and Weis (1989)
acute	rat	oral		2 g/kg		single	decreased urine volume and creatine concentration	Freundt and Weis (1989)
acute	rat	oral	0.2 g/kg			single	no effect	Freundt and Weis (1989)
acute	rat	oral			14,800 mg/kg	single	death	Tolstopiatova et al. (1987)
acute	rat	oral			13,000-32,000 mg/kg	single	death	Laug et al. (1939); Smyth et al. (1941).
acute	rat	intraperitoneal injection			7,700 mg/kg	single	N/R	Patty (1982)
acute	rat	subcutaneous injection			18,800 mg/kg	single	N/R	Patty (1982)

Table A-6. Toxicity of DEG to Mammalian Species

Study Type	Species	Route	NOAEL/ NOAEC	LOAEL/ LOAEC/ LPTD	LD50/ LC50	Duration/ Exposure	Endpoint	Reference
acute	rat	intraperitoneal injection			11,000 mg/kg (LPLD)	single	convulsions and death	Kraul et al (1991)
acute	rat	intraperitoneal injection		2,800 mg/kg		single	weak effects on kidney	Kraul et al (1991)
acute	mouse	inhalation	saturated vapour			8 hour	no mortality	Deichmann (1969)
acute	guinea-pig	dermal	30% solution in ethylene glycol			4 hour	non-irritant	Ishihara and Ikeda (1979)
acute	guinea-pig	dermal	neat DEG			2-14 days	non-irritant	Loeser (1954)
acute	human	dermal			6,200 mg/kg of substance (LPLC)	unspecified	kidney failure, liver damage, CNS effects, death of 5 patients following the use of DEG as a solvent for a drug used in serious burn cases.	Cantarell et al. (1987)
acute	human	dermal	20% in petrolatum			48 hour	non-irritant	Meneghini et al. (1971)
acute	rabbit	dermal		500 mg		single	mild irritation	Deichmann (1969)
acute	rabbit	ocular	0.1-0.5 ml neat DEG			single	non-irritant	Carpenter and Smyth (1946)
acute	rabbit	dermal	10% aqueous solution	neat DEG		single	mild irritation	Guillot et al. (1982)
acute	rabbit	ocular	0.1-0.5 ml neat DEG			single	non-irritant	Guillot et al. (1982)
acute	rabbit, dog, cat	ocular	0.1-0.5 ml neat DEG			single	non-irritant	Loeser (1954)
acute	rabbit, dog, rat	dermal	neat DEG			single	non-irritant to oral mucosa	Loeser (1954)
acute	human	inhalation		DEG in cigarettes		2-4 weeks	allergic dermatitis	Newman (1938)

Table A-6. Toxicity of DEG to Mammalian Species

Study Type	Species	Route	NOAEL/ NOAEC	LOAEL/ LOAEC/ LPTD	LD50/ LC50	Duration/ Exposure	Endpoint	Reference
Sub-Chronic and Chronic								
chronic	rat	oral	50 mg/kg/day	100 mg/kg/day		225 days	marginal increase in urinary oxalate	BIBRA (1976)
sub-chronic	hamster	oral	2% DEG in drinking water, about 3 g/kg bw/day	3% DEG in drinking water, about 4.5 g/kg bw/day		probably 3 weeks	death	Yoshida et al. (1986)
sub-chronic	human	oral		1,300 - 4,000 mg/kg		unknown	DEG in pharmaceuticals has led to the deaths of 71 adults and 150 children in three incidents	Calvery and Klumpp (1939); Geiling an Cannon (1938); Bowie and Mackenzie (1972); Renwick and Cameron (1992)
sub-chronic	mouse	oral		50 mg/kg bw/day		14-17 week	effects on blood clotting and immune response	Huber et al. (1986)
sub-chronic	mouse	oral	5,200 mg/kg bw/day			15-18 weeks	no overt toxic effects	Morrissey et al. (1988)
sub-chronic	mouse	oral		11,200 mg/kg bw/day		day 6-13 of pregnancy	2/50 died	Hardin et al (1987)
sub-chronic	mouse	oral	1,250 mg/kg bw/day	5,000 mg/kg bw/day		day 6-15 of pregnancy	reduced weight gain and kidney effects	Bates et al. (1991)
sub-chronic	rabbit, guinea-pig, cat, dog	oral		1,000-5,000 mg/kg bw/day		28 days	kidney and liver damage	Hanzlik et al. (1939)
sub-chronic	rat	oral		1,000-2,000 mg/kg/day		11 days-3 months	kidney and liver damage	Bomman (1955, 1954a,b); Loeser (1954); Tolstopiatova (1987).
sub-chronic	rat	oral	200 mg/kg/day			90 days	no effect on kidney weight or urinary biochemical parameters	Freundt and Weis (1989)
reproduction	rat	oral		1/50 LD_{50}		days 0-19 of pregnancy	soviet study: increase in foetal abnormalities involving CNS, eyes, urogenital and skeletal system	Barilyak (1989)
sub-chronic	rat	oral		3 mg/kg bw/day		30 days	soviet study: effects on sperm and testes in males (uncertain whether DEG was in the mix of glycols used)	Byshovetets et al. (1987)
sub-chronic	rat	oral		15 mg/kg bw/day		30 days	soviet study: effects on oestrus cycle in females (uncertain whether DEG was in the mix of glycols used)	Byshovetets et al. (1987)

Table A-6. Toxicity of DEG to Mammalian Species

Study Type	Species	Route	NOAEL/ NOAEC	LOAEL/ LOAEC/ LPTD	LD50/ LC50	Duration/ Exposure	Endpoint	Reference
sub-chronic	rabbit	dermal		50% solution in propylene glycol		100 days, daily application	slight microscopic changes	Rantuccio et al. (1979)
Reproduction and Development								
reproduction	mouse	oral	3.1 g/kg bw/day	6.1 g/kg bw/day		continuous exposure in drinking water	mildly toxic to parent animals, reduction in number and size of litters, proportion of pups born alive, and pup weight, increase in fetal abnormalities, decreased fertility.	Williams et al, (1990)
reproduction	mouse	oral	11.2 g/kg bw/day			Days 6-13 of pregnancy	no effect on number of viable litters, pup birth weight, or pup survival at day 3 of life	Hardin et al (1987)
reproduction	mouse	oral	5 g/kg bw/day	10 g/kg bw/day		Days 6-15 of pregnancy	reduced fetal weight	Bates et al. (1991)
reproduction	mouse	oral		343 gm/kg		multigeneration	maternal effects - parturition; effects on number and sex ratio of young.	Williams et al, (1990)
reproduction	mouse	oral		343 gm/kg		multigeneration	effects on female fertility index and effects on newborn	Williams et al, (1990)
reproduction	rat	oral	2.2 g/kg bw/day			12 weeks	normal fertility, normal offspring	Wegener (1953)
reproduction	rat	oral	0.5 g/kg bw/day	1.5 g/kg bw/day		2 generations	Increased kidney weights in parental and first generation	Rodwell et al. (1987)
reproduction	hamsters	intraperitoneal injection	1.8 g/kg bw	2.5 g/kg bw		day 8 of pregnancy	fetal malformations, embryo and fetotoxicity.	Renwick and Cameron (1992)
Carcinogenicity and Genotoxicity								
cancer	rat	oral		1.5 g/kg bw/day		2 year	bladder tumors, mainly benign, seen in about half of the treated animals	Fitzhugh and Nelson (1946)
cancer	rat	oral	2% DEG following exposure to a known bladder carcinogen			32 weeks	exposure to DEG did not increase the incidence of tumors over that with the known bladder carcinogen alone.	Masui et al. (1988)
cancer	rat	oral		4% DEG in diet		ns	bladder tumor developed in one male but no females	Weil et al. (1965)
cancer	mice	dermal	2 drops neat DEG on skin			3x per week for 2 years	no convincing effects of skin carcinogenicity	Vasil'eva et al. (1971)
cancer	mouse	injection	1.5 g/kg bw/ week			70 weeks	no treatment-related tumors	Dunkelberg (1987)

Table A-6. Toxicity of DEG to Mammalian Species

Study Type	Species	Route	NOAEL/ NOAEC	LOAEL/ LOAEC/ LPTD	LD50/ LC50	Duration/ Exposure	Endpoint	Reference
cancer	mouse	inhalation		4-5 mg/m^3		2 hr/day for 6-7 months	10 out of 16 mice developed malignant mammary tumors in 18 months. The incidence of this common tumour in the control group is not recorded.	Sanina (1968)
cancer	human	occupational exposure				nd	increased risk of brain cancer at a US petrochemical plant associated with exposure to a number of chemicals including DEG, though the investigators were unconvinced of a causal relationship with DEG	Leffingwell et al. (1989)
cancer	human	occupational exposure				nd	increased risk of brain cancer at a US petrochemical plant associated with exposure to a number of chemicals including DEG, though the investigators were unconvinced of a causal relationship with DEG	Austin and Schnatter (1983)
cancer	human	occupational exposure				nd	No cancers seen in a group of 90 workers exposed to DEG for 1-9 years.	Telegina et al. (1971)
genotoxicity	hamster	oral		5,000 mg/kg		12 week	equivocal results	Yoshida et al. (1986)
genotoxicity	hamster	oral		3 g/kg		3 week	equivocal results	Yoshida et al. (1986)
genotoxicity	hamster	oral		7,500 mg/kg bw		single	equivocal results	Yoshida et al. (1986)
genotoxicity	hamster	intraperitoneal injection		1.25 g/kg bw		single	slight increase in incidence of chromosomal damage	Yoshida et al. (1986)
genotoxicity	bacteria	other				single	no evidence of mutagenicity in Ames tests using the bacterium Salmonella typhimurium in the presence or absence of a liver metabolic activation system.	Pfeiffer and Dunkelberg (1980); Yoshida et al. (1986); Zeiger et al. (1987).

Notes:

NV = not reported in the abstract and not verified in this literature search
dw = drinking water
LPLD = lowest published lethal dose
LPTD = lowest published toxic dose
NOAEL/NOAEC = no observed adverse effect level/concentration
LOAEL/LOAEC = lowest observed adverse effect level/concentration
LD50/LC50 = lethal dose/concentration for 50% kill

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APPENDIX B

TEG DEGRADATION AND TOXICITY DATA

Table B-1 Summary of Available Data on TEG Biodegradation

Table B-2 Toxicity of TEG to Freshwater Aquatic Life

Table B-3 Toxicity of TEG to Marine Aquatic Life

Table B-4 Toxicity of TEG to Terrestrial Plants

Table B-5 Toxicity of TEG to Terrestrial Invertebrates

Table B-6 Toxicity of TEG to Mammalian Species

Table B-1. Summary of Available Information on TEG Biodegradation

Test Method	Test Duration	Aerobic/ Anaerobic	Initial Compound Concentration	% Removed	Inoculum or Medium	Interpreted Half Life	Rates / Comments	Reference
Studies Conducted under Unamended Conditions								
batch cultures; IEC analysis	98 days	aerobic	2,100 mg/kg	100%	contaminated soil groundwater slurry	175 days	Complete degradation achieved after phosphate amendment. Interpreted half life is based on 64 days of unamended degradation	Mrklas et al. (2004)
batch cultures; GC/MS analysis	35 days	aerobic	100 ppm	50%	distilled water	35 days	Similar rates for aerobic, anaerobic, and abiotic. Degradation interpreted to be abiotic in all cases.	Kaplan et al. (1982)
respirometry	62 days	aerobic	200 mg/kg	100%	Alberta Soil	11 days	lag period: 3.7 days, hyperbolic kinetics; 25C	Sorensen et al. (2000)
respirometry	62 days	aerobic	1,000 mg/kg	72%	Alberta Soil	20 days	lag period: 4.4 days, hyperbolic kinetics; 25C	Sorensen et al. (2000)
respirometry	62 days	aerobic	200 mg/kg	93%	New Mexico Soil	11 days	lag period: 2.4 days, hyperbolic kinetics; 25C	Sorensen et al. (2000)
respirometry	62 days	aerobic	1,000 mg/kg	32%	New Mexico Soil	97 days	lag period: 13.1 days, hyperbolic kinetics; 25C	Sorensen et al. (2000)
respirometry	62 days	aerobic	200 mg/kg	53%	Louisiana Soil	58 days	lag period: 13.1 days, arithmentc kinetics; 25C	Sorensen et al. (2000)
respirometry	62 days	aerobic	1,000 mg/kg	24%	Louisiana Soil	131 days	lag period: 1.9 days, arithmetic kinetics; 25C	Sorensen et al. (2000)
respirometry	28 days	aerobic	17,700 mg/L COD	9-28%	soil-water-rich glycol slurry	nd	removal based on COD; rich glycol from Texas facility	Sorensen et al. (2000)
respirometry	28 days	aerobic	17,600 mg/L COD	0-18%	soil-water-rich glycol slurry	nd	removal based on COD; rich glycol from Louisiana facility	Sorensen et al. (2000)
Other Studies								
degradability test	5 days	aerobic	nv	nv	nv	nd	"extensive" degradation ofter 5 days	Haines and Alexander (1975)
batch cultures; IEC analysis	98 days	aerobic	2,100 mg/kg	100%	contaminated soil groundwater slurry	25 days	Degradation rate after phosphate amendment	Mrklas et al. (2004)
batch cultures; GC/MS analysis	35 days	both	100 ppm	50%	acclimated sludge and nutrient broth	35 days	Similar rates for aerobic, anaerobic, and abiotic. Degradation interpreted to be abiotic in all cases.	Kaplan et al. (1982)
respirometry	30 days	aerobic	13,150 mg/L	96-100%	N&P Amended soil-water-rich glycol slurry	nd	glycol removal determined directly by chemical analysis; rich glycol from Texas facility	Sorensen et al. (2000)
respirometry	30 days	aerobic	12,760 mg/L	39-100%	N&P Amended soil-water-rich glycol slurry	nd	glycol removal determined directly by chemical analysis; rich glycol from Louisiana facility	Sorensen et al. (2000)

Table B-1. Summary of Available Information on TEG Biodegradation

Test Method	Test Duration	Aerobic/ Anaerobic	Initial Compound Concentration	% Removed	Inoculum or Medium	Interpreted Half Life	Rates / Comments	Reference
respirometry	30 days	aerobic	9,660 mg/L	86%	N&P Amended -water-rich glycol slurry	nd	glycol removal determined directly by chemical analysis; rich glycol from Texas facility	Sorensen et al. (2000)
respirometry	30 days	aerobic	9,660 mg/L	27%	N&P Amended -water-rich glycol slurry	nd	glycol removal determined directly by chemical analysis; rich glycol from Louisiana facility	Sorensen et al. (2000)
degradability test	8 hours	aerobic	333 mg/L	nv	acclimated sludge	nd	slight reduction in BOD/COD	Hatfield (1957)
degradability test	nv	aerobic	nv	nv	TEG-adapted gram negative bacterium	nd	bacterium could grow on TEG, but TEG not utilized if alternative carbon source available	Fincher and Payne (1962)
degradability test	nv	aerobic	nv	nv	adapted cells	nd	TEG consumed 92% of theoretical oxygen consumption, indicating degradation of TEG; oxidation rate decreased TEG>DEG>EG	Fincher and Payne (1962)
degradability test	nv	aerobic	nv	nv	Glucobacter oxydans	nd	positive result for glycol oxidation	Kersters and DeLey (1963)
degradability test	3-14 days	aerobic	2-10 mg/L	nv	river water	nd	biodegdatation in some waters was complete after 4 days at both 4 and 20C, in other waters no measurable degradation occurred in 14 days at either temperature.	Evans and David (1974)
degradability test	nv	aerobic	nv	nv	nv	nd	Degradation by the G17bacterium genus *Alcaligenes* noted	Harada and Nagashima (1975)
degradability test	nv	aerobic	200 mg/L (as COD)	98%	sludge	nd	rapid degradation	Pitter (1976)
degradability test	nv	aerobic	nv	nv	nv	nd	Degraded by bacteria of genus *Acinetobacter* and *Pseudomonas*, but not *Flavobacterium.*	Jones and Watson (1976)
degradability test	nv	aerobic	nv	nv	nv	nd	Degraded by bacteria of genus *Pseudomonas*	Thelu et al. (1980)
degradability test	nv	both	nv	nv	aerobic and anaerobic sludge	nd	Degradation observed, but appeared to be abiotic. Analysis by GC.	Kaplan et al. (1982)
degradability - anaerobic	nv	anaerobic	nv	nv	nv	nd	glycol metabolism by *Desulfovibrio desulphuricans.*	Dwyer and Tiedje (1983)
BOD reduction	5 days	aerobic	nv	1.4-32%	nv	nd	based on theoretical oxygen demand (ThOD)	Verschueren (2001)
BOD reduction	10 days	aerobic	nv	3.7-64%	nv	nd	based on theoretical oxygen demand (ThOD)	Verschueren (2001)
BOD reduction	15 days	aerobic	nv	11.5-77%	nv	nd	based on theoretical oxygen demand (ThOD)	Verschueren (2001)
BOD reduction	20 days	aerobic	nv	17-86%	nv	nd	based on theoretical oxygen demand (ThOD)	Verschueren (2001)

Table B-1. Summary of Available Information on TEG Biodegradation

Test Method	Test Duration	Aerobic/ Anaerobic	Initial Compound Concentration	% Removed	Inoculum or Medium	Interpreted Half Life	Rates / Comments	Reference
degradability	up to 35 days	aerobic	various	up to 98%	various	nd	13 data point s from 6 studies, most indicating significant biodegradation - see Figure 4.3	Verschueren (2001)
inhibition of biodegradation	24 hours	aerobic	4,000 mg/L	no effect	activated sludge	nd	no inhibition on biodegradation at 4,000 mg/L	Verschueren (2001)

[a] Biochemical oxygen demand (BOD) is defined as parts of oxygen consumed per part of compound during degradation. This value is expressed as a percentage of the theoretical (ThOD) oxygen demand.

nv = not reported in the abstract and not verified in the literature search

nd = not determined

Table B-2. Toxicity of TEG to Freshwater Aquatic Life

Biota Type	Scientific Name	Common Name	Study Type	Test Duration	Concentration	Endpoint	Effect	Exposure Type	pH	Temperature	Chemical Analysis	Control Type	Reference
					mg/L					°C			
Primary Data													
vertebrate	Lepomis macrochirus	Bluegill	acute	115 h	60,157	LC50	mortality	flow-through	7.94	25.2	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Lepomis macrochirus	Bluegill	acute	168 h	60,157	LC50	mortality	flow-through	7.94	25.2	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Lepomis macrochirus	Bluegill	acute	91 h	61,000	LC50	mortality	flow-through	7.94	25.2	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Lepomis macrochirus	Bluegill	acute	96 h	61,000	LC50	mortality	flow-through	7.94	25.2	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Lepomis macrochirus	Bluegill	acute	76 h	64,200	LC50	mortality	flow-through	7.94	25.2	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Lepomis macrochirus	Bluegill	acute	67 h	66,300	LC50	mortality	flow-through	7.94	25.2	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Lepomis macrochirus	Bluegill	acute	41 h	69,400	LC50	mortality	flow-through	7.94	25.2	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Lepomis macrochirus	Bluegill	acute	19 h	75,190	LC50	mortality	flow-through	7.94	25.2	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Pimephales promelas	Fathead minnow	acute	96 h	59,900	LC50	mortality	flow-through	7.3	NV	measured	satisfactory	Geiger et al. (1988)
vertebrate	Pimephales promelas	Fathead minnow	acute	96 h	70,200	LC50	mortality	flow-through	7.3	22.2	measured	satisfactory	Geiger et al. (1988)
vertebrate	Pimephales promelas	Fathead minnow	acute	96 h	77,400	LC50	mortality	flow-through	NV	21.7	measured	satisfactory	Geiger et al. (1988)
vertebrate	Pimephales promelas	Fathead minnow	acute	96 h	92,500	LC50	mortality	flow-through	7.91	25.5	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Pimephales promelas	Fathead minnow	acute	168 h	92,500	LC50	mortality	flow-through	7.91	25.5	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Pimephales promelas	Fathead minnow	acute	24 h	95,000	LC50	mortality	flow-through	7.91	25.5	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Pimephales promelas	Fathead minnow	acute	12 h	104,000	LC50	mortality	flow-through	7.91	25.5	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Salvelinus fontinalis	Brook trout	acute	18 h	73,499	LC50	mortality	flow-through	7.59	15.8	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Salvelinus fontinalis	Brook trout	acute	121 h	73,499	LC50	mortality	flow-through	7.59	15.8	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Salvelinus fontinalis	Brook trout	acute	48 h	73,500	LC50	mortality	flow-through	7.59	15.8	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Salvelinus fontinalis	Brook trout	acute	96 h	73,500	LC50	mortality	flow-through	7.59	15.8	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Salvelinus fontinalis	Brook trout	acute	160 h	73,500	LC50	mortality	flow-through	7.59	15.8	measured	satisfactory	Cardwell et al. (1978)
vertebrate	Salvelinus fontinalis	Brook trout	acute	168 h	73,500	LC50	mortality	flow-through	7.59	15.8	measured	satisfactory	Cardwell et al. (1978)
invertebrate	Hyalella azteca	Amphipod	acute	96 h	43,500	LC50	Mortality	Static	7.9	23	measured	satisfactory	Vizon (2006)
Secondary Data													
vertebrate	Pimephales promelas	fathead minnow	acute	70 h	82,000	LC50	mortality	static	NV	25	nominal	satisfactory	Cardwell et al. (1978)
vertebrate	Pimephales promelas	fathead minnow	acute	168 h	82,000	LC50	mortality	static	NV	25	nominal	satisfactory	Cardwell et al. (1978)
vertebrate	Pimephales promelas	fathead minnow	acute	22 h	85,500	LC50	mortality	static	NV	25	nominal	satisfactory	Cardwell et al. (1978)
invertebrate	Chironomus tentans	midge	chronic	48 h	64,000	LC50	mortality	static	NV	NV	nominal	satisfactory	Ziegenfuss et al. (1986)
invertebrate	Daphnia magna	water flea	acute	48 h	39,375	LC50	mortality	static	7.9-8.3	21	nominal	satisfactory	LeBlanc and Surprenant (1983)
invertebrate	Daphnia magna	Water flea	acute	2 d	42,426	EC50	Immobilization	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	acute	48 h	46,500	EC50	Immobilization	static	NV	20-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	acute	48 h	52,400	EC50	Immobilization	static	7.8	22	nominal	satisfactory	Barera and Adams (1983)
invertebrate	Daphnia magna	water flea	acute	24 h	65,250	LC50	mortality	static	7.9-8.3	21	nominal	satisfactory	LeBlanc and Surprenant (1983)

Table B-2. Toxicity of TEG to Freshwater Aquatic Life

Biota Type	Scientific Name	Common Name	Study Type	Test Duration	Concentration	Endpoint	Effect	Exposure Type	pH	Temperature	Chemical Analysi	Control Type	Reference
					mg/L					°C			
invertebrate	Daphnia magna	Water flea	acute	24 h	78,500	EC50	Immobilization	static	NV	20-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	acute	24 h	88,500	EC50	Immobilization	static	7.8	22	nominal	satisfactory	Barera and Adams (1983)
invertebrate	Daphnia magna	Water flea	chronic	28 d	12,375	LOEC	mortality	flow-through	7.3-8	21	nominal	satisfactory	LeBlanc and Surprenant (1983)
invertebrate	Daphnia magna	Water flea	chronic	28 d	12,375	LOEC	reproduction	flow-through	7.3-8	21	nominal	satisfactory	LeBlanc and Surprenant (1983)
invertebrate	Daphnia magna	Water flea	chronic	21 d	15,000	LOEC	Length	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	21 d	21,213	MATC	survival	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	21 d	21,213	MATC	reproduction	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	14 d	30,000	LOEC	survival	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	21 d	30,000	LOEC	survival	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	water flea	chronic	7 d	30,000	LOEC	length	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	21 d	33,911	EC50	Immobilization	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	14 d	39,356	EC50	Immobilization	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	7 d	40,538	EC50	Immobilization	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	7 d	42,426	MATC	survival	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	14 d	42,426	MATC	survival	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	7 d	42,426	MATC	survival	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	14 d	42,426	MATC	reproduction	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	7 d	60,000	LOEC	survival	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	water flea	acute	48 h	52,400	LC50	mortality	static	NV	NV	nominal	satisfactory	Ziegenfuss et al. (1986)
other	Microcystis aeruginosa	blue-green algae	chronic	8 d	3,600	LOEC	growth	static	7	27	nominal	satisfactory	Bringmann and Kuhn (1978b)
Unacceptable Data (Based on Unverifiable Control Information)													
other	Anacystis aeruginosa	blue-green algae	NV	NV	3,600	NV	mortality	static	7	27	nominal	NV	Bringmann and Kuhn (1978d)
other	Anacystis aeruginosa	blue-green algae	chronic	8 d	3,600	NV	growth	static	NV	27	nominal	NV	Bringmann and Kuhn (1978c)
Data Excluded Due to Lack of Effects at Maximum Concention Tested													
invertebrate	Daphnia magna	Water flea	acute	48 h	24,000	NOEC	Immobilization	static	7.8	22	nominal	satisfactory	Barera and Adams (1983)
invertebrate	Daphnia magna	Water flea	acute	48 h	24,000	NOEC	Immobilization	static	NV	20-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	7 d	30,000	NOEC	survival	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	14 d	15,000	NOEC	survival	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	21 d	15,000	NOEC	survival	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	7 d	15,000	NOEC	Length	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	21 d	7,500	NOEC	Length	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	14 d	>15,000	NOEC	reproduction	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)
invertebrate	Daphnia magna	Water flea	chronic	21 d	>15,000	NOEC	reproduction	renewal	7.2-8.5	21-23	nominal	satisfactory	Adams and Heidolph (1985)

Table B-2. Toxicity of TEG to Freshwater Aquatic Life

Biota Type	Scientific Name	Common Name	Study Type	Test Duration	Concentration	Endpoint	Effect	Exposure Type	pH	Temperature	Chemical Analysis	Control Type	Reference
					mg/L					°C			
invertebrate	Daphnia magna	water flea	acute	24 h	>10,000	LC50	mortality	static	7.6-7.7	20-22	nominal	NV	Bringmann and Kuhn (1977b)
invertebrate	Daphnia magna	water flea	acute	24 h	>10,000	EC50	behaviour	NV	8	NV	NV	NV	Bringmann and Kuhn (1982)
other	Entosiphon sulcatum	flagellate euglenoid	chronic	72 h	>1,000	NV	growth	static	6.9	25	nominal	NV	Bringmann and Kuhn (1978a)
other	Entosiphon sulcatum	flagellate euglenoid	NV	NV	>10,000	NV	growth	NV	NV	NV	NV	NV	Bringmann and Kuhn (1979)
other	Uronema parduczi	ciliate	acute	20 h	>10,000	NV	growth	NV	6.9	NV	NV	NV	Bringmann and Kuhn (1980b)
other	Chilomonas paramecium	cryptomonad	NV	NV	>10,000	NV	growth	NV	NV	NV	NV	NV	Bringmann and Kuhn (1981)
other	Entosiphon sulcatum	flagellate euglenoid	NV	NV	>10,000	NV	growth	NV	NV	NV	NV	NV	Bringmann and Kuhn (1981)
other	Uronema parduczi	ciliate	NV	NV	>10,000	NV	growth	NV	NV	NV	NV	NV	Bringmann and Kuhn (1981)
other	Entosiphon sulcatum	flagellate euglenoid	chronic	72 h	>10,000	NV	growth	static	NV	25	nominal	NV	Bringmann and Kuhn (1980a)
other	Chilomonas paramecium	cryptomonad	chronic	48 h	>10,000	NV	growth	NV	6.9	20	nominal	NV	Bringmann et al. (1980)
plant/alga	Scenedesmus quadricauda	green algae	NV	NV	>10,000	LOEC	growth	static	NV	NV	nominal	NV	Bringmann and Kuhn (1978b)
plant/alga	Scenedesmus quadricauda	green algae	NV	NV	>10,000	NV	growth	NV	NV	NV	NV	NV	Bringmann and Kuhn (1979)
plant/alga	Chlorococcales	green algae order	acute	24 h	>1,000	EC10	imilation efficie	static	NV	NV	NV	NV	Krebs (1991)
plant/alga	Scenedesmus quadricauda	green algae	NV	NV	>10,000	NV	growth	static	7	27	nominal	NV	Bringmann and Kuhn (1977a)
plant/alga	Scenedesmus quadricauda	green algae	NV	NV	>10,000	NV	mortality	static	7	27	nominal	NV	Bringmann and Kuhn (1978d)
plant/alga	Scenedesmus quadricauda	green algae	chronic	7 d	>10,000	NV	growth	static	NV	27	nominal	NV	Bringmann and Kuhn (1980a)
plant/alga	Scenedesmus quadricauda	green algae	chronic	8 d	>10,000	NV	growth	static	NV	27	nominal	NV	Bringmann and Kuhn (1978c)
vertebrate	Carassius auratus	goldfish	acute	24 h	>5,000	LC50	mortality	static	7	20	measured	satisfactory	Bridie et al. (1979)
vertebrate	Lepomis macrochirus	bluegill	acute	96 h	>10,000	LC50	mortality	static	7.6-7.9	23	nominal	NV	Dawson et al. (1977)
vertebrate	Leuciscus idus melanotus	carp	acute	48 h	>10,000	LC50	mortality	NV	NV	NV	NV	NV	Juhnke and Luedemann (1978)

Notes:

NV = not reported in the abstract and not verified in this literature search

Table B-3. Toxicity of TEG to Marine Aquatic Life

Biota Type	Scientific Name	Common Name	Study Type	Test Duration	Concentration	Endpoint	Effect	Exposure Type	pH	Temperature	Salinity	Chemical Analysi	Control Type	Reference
					mg/L					°C	ppt			
Primary Data														
invertebrate	Americamysis bahia	opossum shrimp	chronic	23 d	1,000	NOEC	mortality	flow-through	7.81	25	20	m	s	Montgomery et al. (1985)
invertebrate	Americamysis bahia	opossum shrimp	chronic	23 d	1,000	NOEC	reproduction	flow-through	7.81	25	20	m	s	Montgomery et al. (1985)
vertebrate	Cyprinodon variegatus	sheepshead minnow	acute	28 d	8	BCF	accumulation	flow-through	NV	30	25.2	m	s	Goodman et al. (1978)
vertebrate	Cyprinodon variegatus	sheepshead minnow	acute	28 d	8	NV	length	flow-through	NV	30	25.2	m	s	Goodman et al. (1978)
vertebrate	Cyprinodon variegatus	sheepshead minnow	acute	28 d	8	NV	survival	flow-through	NV	30	25.2	m	s	Goodman et al. (1978)
vertebrate	Menidia beryllina	inland silverside	chronic	28 d	<0	NV	dry weight	flow-through	7.5	23-26	30	m	s	Thursby and Berry (1987a)
vertebrate	Menidia beryllina	inland silverside	chronic	28 d	<0	NV	mortality	flow-through	7.5	23-26	30	m	s	Thursby and Berry (1987a)
vertebrate	Menidia beryllina	inland silverside	acute	96 h	<=56	NV	survival	flow-through	7.5	25	30	m	s	Thursby and Berry (1987a)
vertebrate	Menidia peninsulae	tidewater silverside	chronic	28 d	40	NV	growth	flow-through	7.73	25	20	m	s	Montgomery et al. (1985)
vertebrate	Menidia peninsulae	tidewater silverside	chronic	28 d	230	NV	growth	flow-through	7.73	25	20	m	s	Montgomery et al. (1985)
vertebrate	Menidia peninsulae	tidewater silverside	chronic	28 d	230	NV	mortality	flow-through	7.73	25	20	m	s	Montgomery et al. (1985)
vertebrate	Menidia peninsulae	tidewater silverside	chronic	28 d	1,500	NV	mortality	flow-through	7.73	25	20	m	s	Montgomery et al. (1985)
Secondary Data														
invertebrate	Americamysis bahia	Opossum shrimp	acute	24-96 h	563	NV	survival	renewal	NV	24.8	31	n	s	Thursby and Berry (1987a)
invertebrate	Ampelisca abdita	Amphipod	acute	96 h	563	NV	survival	NV	8	NV	NV	n	s	Thursby and Berry (1987a)
invertebrate	Arbacia punctulata	Purple-spined sea urchin	acute	48 h	563	NV	development	static	8	19	30	n	s	Thursby and Berry (1987a)
invertebrate	Arbacia punctulata	purple-spined sea urchin	acute	48 h	563	NV	survival	static	8	19	30	n	s	Thursby and Berry (1987a)
invertebrate	Crassostrea virginica	american or virginia oyster	acute	48 h	563	NV	survival	static	7.7	27.2	32	n	s	Thursby and Berry (1987a)
invertebrate	Dinophilus gyrociliatus	archiannelid	acute	96 h	563	NV	survival	renewal	8	20	32	n	s	Thursby and Berry (1987a)
invertebrate	Palaemonetes pugio	daggerblade grass shrimp	acute	96 h	563	NV	survival	renewal	8	25	29	n	s	Thursby and Berry (1987a)
plant/alga	Laminaria saccharina	tangleweed, brown algae	acute	48 h	563	NV	reproduction	static	NV	14	30	n	s	Thursby and Steele (1987)
vertebrate	Cyprinodon variegatus	sheepshead minnow	acute	96 h	563	NV	survival	renewal	8.1	25	30	n	s	Thursby and Berry (1987b)
vertebrate	Menidia beryllina	inland silverside	acute	48-96 h	563	NV	survival	renewal	NV	25	32-37	n	s	Thursby and Berry (1987b)
vertebrate	Menidia beryllina	inland silverside	acute	24 h	563	NV	survival	renewal	NV	25	32-37	n	s	Thursby and Berry (1987b)
vertebrate	Pleuronectes americanus	winter flounder	acute	72 h	563	NV	survival	static	NV	5	30	n	s	Thursby and Berry (1987b)

Table B-3. Toxicity of TEG to Marine Aquatic Life

Biota Type	Scientific Name	Common Name	Study Type	Test Duration	Concentration mg/L	Endpoint	Effect	Exposure Type	pH	Temperature °C	Salinity ppt	Chemical Analysis	Control Type	Reference
Unacceptable or Unverified Data														
invertebrate	Artemia salina	brine shrimp	acute	24 h	>10,000	LC50	mortality	static	NV	24	NV	n	NV	Price et al. (1974)
vertebrate	Petromyzon marinus	sea lamprey	acute	24 h	5	NV	stress	static	7.5-8.2	13	nv	n	NV	Applegate et al. (1957)
vertebrate	Menidia beryllina	inland silverside	acute	96 h	>10,000	LC50	mortality	static	7.6-7.9	20	NV	n	NV	Dawson et al. (1977)

Notes:

NV = not reported in the abstract and not verified in this literature search

chemical analysis: m = measured, n = nominal

control type: c = concurrent; s = satisfactory

Table B-4. Toxicity of TEG to Terrestrial Plants

Scientific Name	Common Name	Effect Measurement	Concentration	Endpoint/ Response	Response Site	Test Duration	Media Type	Application Method	Chemical Analysis	Reference
						days				
Medicago sativa	Alfalfa	Length	7,158	IC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Medicago sativa	Alfalfa	Length	9,660	IC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Medicago sativa	Alfalfa	Dry Mass	6,615	IC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Medicago sativa	Alfalfa	Dry Mass	7,433	IC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Length	7,676	IC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Length	10,953	IC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Dry Mass	4,314	IC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Dry Mass	5,132	IC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Length	5,070	IC25	shoot	21	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Length	5,707	IC25	root	21	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Dry Mass	1,924	IC25	shoot	21	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Dry Mass	2,141	IC25	root	21	artificial soil	spiked	Y	Stantec (2006)

Notes:

values presented here are nominal - not corrected for analytical recovery.

Table B-5. Toxicity of TEG to Terrestrial Invertebrates

Scientific Name	Common Name	Effect Measurement	Concentration	Endopint/Response	Test Duration	Media Type	Application Method	Chemical Analysis	Reference
					days				
Eisenia andrei	Earthworm	# progeny	9,418	IC25	63	artificial soil	spiked	Y	Stantec (2006)
Eisenia andrei	Earthworm	progeny wet mass	7,919	IC25	63	artificial soil	spiked	Y	Stantec (2006)
Eisenia andrei	Earthworm	progeny dry mass	7,528	IC25	63	artificial soil	spiked	Y	Stantec (2006)
Folsomia candida	Springtail	adult survival	27,352	LC50	28	artificial soil	spiked	Y	Stantec (2006)
Folsomia candida	Springtail	# progeny	13,701	IC25	28	artificial soil	spiked	Y	Stantec (2006)

Notes:

values presented here are nominal - not corrected for analytical recovery.

IC25/LC25 values presented where available, otherwise IC50/LC50 presented

Table B-6. Toxicity of TEG to Mammalian Species

Study Type	Species	Route	NOAEL/ NOAEC	LOAEL/ LOAEC/ LPTD	LD50/ LC50	Duration/ Exposure	Endpoint	Reference
Acute								
acute	rat	oral		50% TEG in		single	irritation to digestive tract	Smyth et al. (1941)
acute	rat guinea-pig	oral			8.8-22 g/kg	single	sluggish behaviour (assumed CNS depression); kidney damage; Soviet study found effects on vascular system, kidney, liver, and brain)	Latven and Molitor (1939); Smyth et al. (1941); Tolstopiatova et al. (1987)
acute	rat	oral			21 g/kg	20 days	soviet study	Tolstopiatova et al. (1987)
acute	human	inhalation	2-4 mg/m^3			4-42 days, continuous or intermittent (8 hour per day)	used to control airborne bacteria in hospital wards; no apparent adverse effects	Bigg et al. (1945); Hamburger et al. (1945); Puck et al. (1945)
acute	rat	intramuscular			8.4 g/kg (LPLD)	single	Nutritional and Gross Metabolic: Body temperature decrease	Lauter and Vrla (1940)
acute	mouse	intravenous			6.5 g/kg	single	N/R	Latven and Molitor (1939)
acute	mouse	subcutaneous			8.8 g/kg	single	N/R	Latven and Molitor (1939)
acute	rabbit	dermal	neat TEG			24 hour	non-irritant	Guillot et al. (1982); Latven and Molitor (1939)
acute	rabbit	dermal			>20 mL/kg	single	N/R	Deichmann (1969.)
acute	human	dermal	20% TEG in petrolatum			48 hour, repeated 5x	no irritation or sensitization	Kligman (1976)
acute	human	dermal	unspecified			unspecified	negligible irritation	Patty, 1981
acute	rabbits	ocular	0.1 ml 10% TEG in water	0.1 ml neat TEG			mild irritation	Guillot et al. (1982)
acute	rabbits	ocular		0.5 ml neat TEG			mild reddening and swelling	Latven and Molitor (1939)
acute	rabbit	ocular		500 mg		single	mild irritation	Carpenter and Smyth (1946)
acute	human	ocular		neat TEG		single	smarting, transient effects on cornea, no permanent damage	Grant (1974)

Table B-6. Toxicity of TEG to Mammalian Species

Study Type	Species	Route	NOAEL/ NOAEC	LOAEL/ LOAEC/ LPTD	LD50/ LC50	Duration/ Exposure	Endpoint	Reference
Sub-Chronic and Chronic								
chronic	rat	oral	2 g/kg/d (in diet)			2 years	no effects on mortality, body weight, blod and urine composition, gross and microscopic appearance of major organs, including testis	Fitzhugh and Nelson (1946)
chronic	rat	oral	3 g/kg/d (in dw)			13 months	no effects on mortality, body weight, blod and urine composition, gross and microscopic appearance of major organs, including testis	Robertson et al. (1947)
chronic	rat	oral	50 mg/kg/d	500 mg/kg/d		6 months	soviet study :mild tissue changes in liver and kidney	Tolstopyatova et al. (1987)
chronic	monkey	oral		300 mg/kg/d		14 months	slight reduction in growth and in the numbers of white blood cells, no effect on urine composition, or in kidney, spleen, or bone marrow	Robertson et al. (1947)
chronic	human	occupational inhalation	unspecified			unspecified	no overt toxicity	Patty (1981); Robertson et al. (1947)
chronic	rat	inhalation	4 mg/m3 (~5 mg/kg /d)			13 months	reproduction appeared normal, though no detailed examination appear to have been made.	Robertson et al. (1947)
chronic	monkey	inhalation	2-3 mg/m^3			10 months	no effect	Robertson et al. (1947)
chronic	monkey	inhalation		4 mg/m3; (~3 mg/kg /d)		13 months	slightly reduced body weight, no other abnormalities in blood, urine, or a microscopic examination of a limited rage of tissues.	Robertson et al. (1947)
chronic	monkey	inhalation	2-3 mg/m^3			10 months	no effect on body weight; no other abnormalities in blood, urine, or a microscopic examination of a limited rage of tissues.	Robertson et al. (1947)
chronic	rat	inhalation	4 mg/m3 (~5 mg/kg /d)			3-13 months	no effect on the composition of blood or urine, on body weight, or on the microscopic appearance of lungs, liver, kidney, or spleen.	Robertson et al. (1947)
sub-chronic	rat	subcutaneous	2.2 g/kg/d	4.5 g/kg/d		4 week	slight abnormalities of blood composition including decreased haemoglobin and inflammation at the injection site	Stenger et al. (1968)
sub-chronic	rat	subcutaneous		1.1 g/kg/d		4 week	elevated urea nitrogen in blood, suggesting possible kidney	Stenger et al. (1968)
sub-chronic	rat (juvenile)	oral	3.7 g/kg/d (in dw)			30 days	no overt toxicity	Lauter and Vrla (1940)

Table B-6. Toxicity of TEG to Mammalian Species

Study Type	Species	Route	NOAEL/ NOAEC	LOAEL/ LOAEC/ LPTD	LD50/ LC50	Duration/ Exposure	Endpoint	Reference
sub-chronic	rat (adult)	oral	2.3 g/kg/d (in dw)		3.7 g/kg/day (in dw)	30 days	no overt toxicity at lower dose, deaths observed at higher dose. (Commercial grade TEG used, toxicity may have been due to an impurity).	Lauter and Vrla (1940)
sub-chronic	rat	oral		1/5,000 to 1/50 of LD_{50}		2-6 months	soviet study: testes damage and adverse effects on sperm of rats (the same laboratory reported a rat oral LD_{50} of 21 g/kg bw (Tolstopyatova et al., 1987).	Byshovets et al. (1987)
sub-chronic	rat	oral		1 g/kg/d		20 days	soviet study :CNS, liver, kidney effects on females rats	Tolstopiatova et al. (1987)
sub-chronic	mouse	oral		11.3 g/kg/d		day 6-15 of pregnancy	increased kidney weight, clinical signs of toxicity	U.S. EPA (1990)
sub-chronic	mouse	oral	3.3 g/kg/d	6.8 g/kg/d		14 weeks	liver weight increases	Bossert et al. (1992)
sub-chronic	monkey	inhalation		4 mg/m^3 ("super-saturated")		3 months	browning of facial skin (bactericidal action of TEG may have promoted a parasitic infection)	Robertson et al. (1947)
sub-chronic	human	inhalation	2.5-3 mg/m^3			6 weeks intermittent	no evidence of irritation to respiratory tract (~8 hours/day assumed)	Bigg et al. (1945)
sub-chronic	rabbit	dermal	neat TEG			covered contact renewed daily for 6 weeks	non-irritant	Guillot et al. (1982)
sub-chronic	human	dermal	unspecified			unspecified	"prolonged contact may cause maceration of the skin"	Patty, 1982
Reproduction and Developmental								
reproduction	rat, mouse	subcutaneous	2.2 g/kg/d			throughout pregnancy	no increase in fetal malformations	Stenger et al. (1968)
reproduction	rabbit	subcutaneous	1.1 g/kg/d			day 7 to 16 of pregnancy	no increase in fetal malformations	Stenger et al. (1968)
reproduction	rat	oral		103 gm/kg		6-15 day pregnant	effects on fetus - musculoskeletal system	U.S. EPA (1990)

Table B-6. Toxicity of TEG to Mammalian Species

Study Type	Species	Route	NOAEL/ NOAEC	LOAEL/ LOAEC/ LPTD	LD50/ LC50	Duration/ Exposure	Endpoint	Reference
reproduction	rat	oral	3 g/kg/d in dw			13 months continuous breeding study	no overt effect on reproduction, though no detailed examinations appear to have been made	Robertson et al. (1947)
reproduction	rat	oral		4.5 g/kg/d		day 1-21 of pregnancy	some sign of fetotoxicity but no increase in foetal malformations	Stenger et al. (1968)
reproduction	rat	oral	1/500 LD50 (~42 mg/kg/d)	1/50 LD50 (420 mg/kg/d)		day 1-19 of pregnancy	soviet study: increase in a range of fetal abnormalities involving a number of organ systems	Barilyak (1989)
reproduction	mouse	oral		56,370 mg/kg		6-15 day pregnant	effects on fetus - musculoskeletal system	U.S. EPA (1990)
reproduction	mouse	oral	0.3% in dw (680 mg/kg/d)	1.5% in dw (3.4 g/kg/d)		14 week	slightly reduced pup weight, no impairment of reproductive efficiency of first generation at 80 days	Bossert et al (1992)
reproduction	mouse	oral	1.5% in dw (3.4 g/kg/d)	3.0 % in dw (6.8 g/kg/d)		14 week	increased liver weight in parental group	Bossert et al (1992)
reproduction	mouse	oral	0.6 g/kg/d	5.6 g/kg/d		day 6-15 of pregnancy	reduced foetal weight, reduced ossification, and increased skeletal variations	U.S. EPA (1990)
reproduction	monkey	oral		0.3 g/kg/d		unspecified period during pregnancy	overtly normal infant died at 2 months. Not thought to be treatment-related.	Robertson et al. (1947)
Carcinogenicity and Genotoxicity								
cancer	rat	oral	4% in diet (~2 g/kg/d)			2 year	no evidence of carcinogenicity (only 12 male rats exposed per dose level)	Fitzhugh and Nelson, (1946)
genotoxicity	rat	oral		2 g/kg/d		2-6 months	soviet study: sperm abnormalities	Byshovets et al. (1987)

Table B-6. Toxicity of TEG to Mammalian Species

Study Type	Species	Route	NOAEL/ NOAEC	LOAEL/ LOAEC/ LPTD	LD50/ LC50	Duration/ Exposure	Endpoint	Reference
genotoxicity	rat	oral		1/50 LD50 (420 mg/kg/d)		single	soviet study: dose administered to male rats that were then mated with untreated females. increased number of early fetal deaths	Barilyak (1989); Byshovets et al. (1987)
genotoxicity	rat	oral		1/5 LD50 (4.2 g/kg/d)		single	soviet study: chromosome damage in bone marrow cells of male rats	Barilyak (1989)
genotoxicity	bacteria	Ames Test				not specified	compound was reported to be mutagenic (no further information available)	NTP (1991)

Notes:

NV = not reported in the abstract and not verified in this literature search

dw = drinking water

LPLD = lowest published lethal dose

LPTD = lowest published toxic dose

NOAEL/NOAEC = no observed adverse effect level/concentration

LOAEL/LOAEC = lowest observed adverse effect level/concentration

LD_{50}/LC_{50} = lethal dose/concentration for 50% kill

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APPENDIX C

TREG
DEGRADATION AND TOXICITY DATA

Table C-1 **Summary of Available Data on TREG Biodegradation**
Table C-2 **Toxicity of TREG to Freshwater Aquatic Life**
Table C-3 **Toxicity of TREG to Marine Aquatic Life**
Table C-4 **Toxicity of TREG to Mammalian Species**

Table C-1. Summary of Available Data on TREG Biodegradation

Test Method	Test Duration	Initial Compound Concentration	% Removed	Inoculum or Medium	Rates / Comments	Reference
degradability test	5 days	nv	nv	nv	"extensive" degradation ofter 5 days	Haines and Alexander (1975)
degradability test	nv	nv	nv	nv	Degradation by the bacterium genus *Alcaligenes* noted	Harada and Nagashima (1975)
degradability - anaerobic	nv	nv	nv	nv	glycol metabolism by *Desulfovibrio desulphuricans*.	Dwyer and Tiedje (1983)
BOD reduction	10 days	nv	22%	nv	based on theoretical oxygen demand (ThOD)	Verschueren (2001)

Biochemical oxygen demand (BOD) is defined as parts of oxygen consumed per part of compound during degradation. This value is expressed as a percentage of the theoretical (ThOD) oxygen demand.

nv = not reported in the abstract and not verified in the literature search

Table C-2. Toxicity of TREG to Freshwater Aquatic Life

Biota Type	Scientific Name	Common Name	Study Type	Test Duration	Concentration	Endpoint	Effect	Exposure Type	pH	Temperature	Chemical Analysis	Control Type	Reference
					mg/L					°C			
Unacceptable or Unverified Data													
vertebrate	Petromyzon marinus	Sea lamprey (larvae, 8-13 cm)	acute	24 h	5	NV	stress	static	7.5-8.2	13	nominal	NV	Applegate et al. (1957)

Table C-3. Toxicity of TREG to Marine Aquatic Life

Biota Type	Scientific Name	Common Name	Study Type	Test Duration	Concentration mg/L	Endpoint	Effect	Exposure Type	pH	Temperature °C	Salinity ppt	Chemical Analysis	Control Type	Reference
Unacceptable or Unverified Data														
invertebrate	*Artemia salina*	Brine shrimp	acute	24 h	10,000	LC50	mortality	static	NV	24	NV	n	NV	Price et al. (1974)

Table C-4. Toxicity of TREG to Mammalian Species

Study Type	Species	Route	NOAEL/ NOAEC	LOAEL/ LOAEC/ LPTD	LD50/ LC50	Duration/ Exposure	Endpoint	Reference[a]
Acute								
acute	rat	oral			32.77 g/kg	single	NV	Smyth et al. (1941)
acute	rat	oral			34 g/kg	single	soviet study: liver and kidney	Tolstopiatova et al. (1987)
acute	guinea-pig	oral			18.75 g/kg	single	soviet study: liver and kidney	Tolstopiatova et al. (1987)
acute	rat	inhalation	"essentially saturated vapours"			8 hour	no significant adverse effects	Patty (1982)
acute	rabbit	ocular		500 mg		single	minimal irritation (scored 1 for irritation on a scale of 1 to 10)	Carpenter and Smyth (1946)
Sub-Chronic and Chronic								
sub-chronic	rat	oral		140,000 mg/kg/d		20 day- intermittent	soviet study: effects on CNS, liver, kidney, bladder	Bandman et al. (1994)
sub-chronic	rat	oral		1,000 mg/kg		10-20 days	soviet study: CNS, liver and kidney effects	Tolstopiatova et al. (1987)
Reproductive and Developmental								
reproductive	rat (female)	oral	68 mg/kg/d	680 mg/kg/d		days 1-19 of pregnancy	soviet study: increased foetal death and malformation rate (CNS, urogenital, and skeletal system). No information on maternal toxicity	Byshovets et al. (1987)
reproduction	rat	oral		200 mg/kg (LPTD)		1 day male	soviet study: reproductive: Effects on fertility: Post-implantation mortality (e.g., dead and/or resorbed implants per total number of implants)	Bandman et al. (1994)
reproduction	rat	oral		6.8 mg/kg/d		2-6 months	soviet study: testes damage and sperm effects	Byshovets et al. (1987)
reproduction	rat (females)	oral		34 mg/kg/d		30 days	soviet study: some changes in oestrus cycle, no further details avilable	Byshovets et al. (1987)
Carcinogenicity and Genotoxicity								
carcinogenicity	human	occupational exposure				NV	increased risk of brain cancer associated with exposure to a wide range of chemicals; investigators were unconvinced that a causal relationship existed with TREG	Leffingwell et al. (1993)
carcinogenicity	human	occupational exposure				NV	This study considered the same group of workers as Leffingwell et al. (1993), but found no association between glycol exposure and cancer risk.	Austin and Schnatter (1983)
genotoxicity	hamster (liver)	cytogenetic analysis		2.5 µg/L		16 hour		Biondi et al., (2002)
genotoxicity	hamster (ovary)	cytogenetic analysis		1.7 mmol/L		3 hour	+enzymatic activation step	Biondi et al., (2002)

Table C-4. Toxicity of TREG to Mammalian Species

Study Type	Species	Route	NOAEL/ NOAEC	LOAEL/ LOAEC/ LPTD	LD50/ LC50	Duration/ Exposure	Endpoint	Reference ^{a}
genotoxicity	hamster (ovary)	cytogenetic analysis		21.6 mmol/L		3 hour	-enzymatic activation step	Biondi et al., (2002)
genotoxicity	rat	oral		2.5 g/kg/d)		2-6 months	soviet study: sperm abnormalities	Byshovets et al. (1987)

Notes:

NV = not reported in the abstract and not verified in this literature search

dw = drinking water

LPLD = lowest published lethal dose

LPTD = lowest published toxic dose

NOAEL/NOAEC = no observed adverse effect level/concentration

LOAEL/LOAEC = lowest observed adverse effect level/concentration

LD_{50}/LC_{50} = lethal dose/concentration for 50% kill

a: numbers in parentheses refer to table of references without authors

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